Experimental Acute Interruption of Portal Vein

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ABSTRACT

Twelve dogs were divided into four groups: Group I-simple interruption of the portal vein for 40 minutes; Group II-interruption of the portal vein combined with interruption of the superior mesenteric artery for 40 minutes; Group III-simple interruption of the portal vein for 60 minutes; Group IV-interruption of the portal vein combined with interruption of the superior mesenteric artery for 60 minutes. The arterial, central venous and portal venous pressures, arterial blood pH, blood sugar, serum glutamic oxalacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) and leucine aminopeptidase values were measured at various intervals during the experiment.

The arterial and central venous pressures fell and the portal venous pressure rose abruptly during and immediately after the interruption. All the dogs were hemodynamically more stable in Groups II and IV than in Groups I and III. The arterial blood pH levels fell to 7.2-7.3 during the interruption and reached their lowest values 60 minutes after release of the interruption. The SGOT, SGPT and leucine aminopeptidase increased after interruption of the portal vein, though these levels Were higher in Groups I and III than in Groups II and IV.

Consequently, it seems safe to interrupt both the portal vein and the superior mesenteric artery during portal vein reconstruction in periampullary cancer of the pancreas.

Key Words: *portal vein interruption*; *superior mesenteric arterial interruption*; *portal venous pressure*

INTRODUCTION

As new operative procedures developed, requirements for portal vein resection to a number of patients with periampullary cancer have

increased. However, it is well known that interruption of the portal vein results in the death of experimental animals within a few hours[1]. There have been many discussions concerning the cause of the deaths in dogs. For decades the cause was ascribed to toxemia, anemia or liver failure[2].

The purpose of this study was to examine the changes in hemodynamics and liver functions during and after interruption of the portal vein and the effect on the two factors of the simultaneous interruption of the superior mesenteric artery.

MATERIALS AND METHODS

Twelve healthy mongrel dogs weighing 7-15 kg were used in this experiment. The dogs were anesthetized by intravenously injecting sodium pentobarbital, 30 mg/kg, and mechanically ventilated via an endotracheal tube during the experiments. The abdomen was opened by an upper median incision without sterilization. The portal vein was separated from the surrounding tissue about 2 cm from the porta hepatis. Interruption of vein, as close to the porta hepatis as possible, was accomplished with Satinsky forceps. The superior mesenteric artery was separated from the surrounding tissue about 2 cm from the origin of the abdominal aorta and interrupted with Satinsky forceps.

The twelve dogs were divided into the following four groups:

Group I-simple interruption of the portal vein for 60 minutes (3 dogs)

Group II-interruption of the portal vein combined with interruption of the superior mesenteric artery for 60 minutes (3 dogs).

Group III-simple interruption of the portal vein for 40 minutes (3 dogs).

Group IV-interruption of the portal vein combined with interruption of the superior mesenteric artery for 40 minutes.

Recordings of the arterial, central and portal venous pressures, and the arterial blood pH, blood sugar, serum glutamic oxalacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) and leucine aminopeptidase (LAP) were made before and during the interruption. The blood pressures and the chemical analyses were followed 30, 60, 90 and 120 min and 60 and 120 min after releasing the forceps, respectively. No fluids or blood transfusions were administered during the experiment.

RESULTS

All of the dogs survived the experiment. After the simple interruption of the portal vein, the surface of the small intestines changed to dark

red and became edematous, but returned to normal immediately after release of the forceps. However, during the interruption of the portal vein combined with superior mesenteric arterial interruption, the small intestines became anemic, contracted and slightly hard.

The mean arterial pressure levels in all groups fell to about 50 % of the pre-interruption levels during the interruption. After release of the forceps, these levels rose remarkably in Groups I and II, though they did not return to the mean arterial pressure. Two hours after release of the forceps the mean arterial pressure in Groups I and II was 74.6 ± 28.1% and 77.8 ± 5.1%, respectively. On the contrary, the level in Group IV was 121.4 ± 43.8% of the pre-interruption level thirty min.

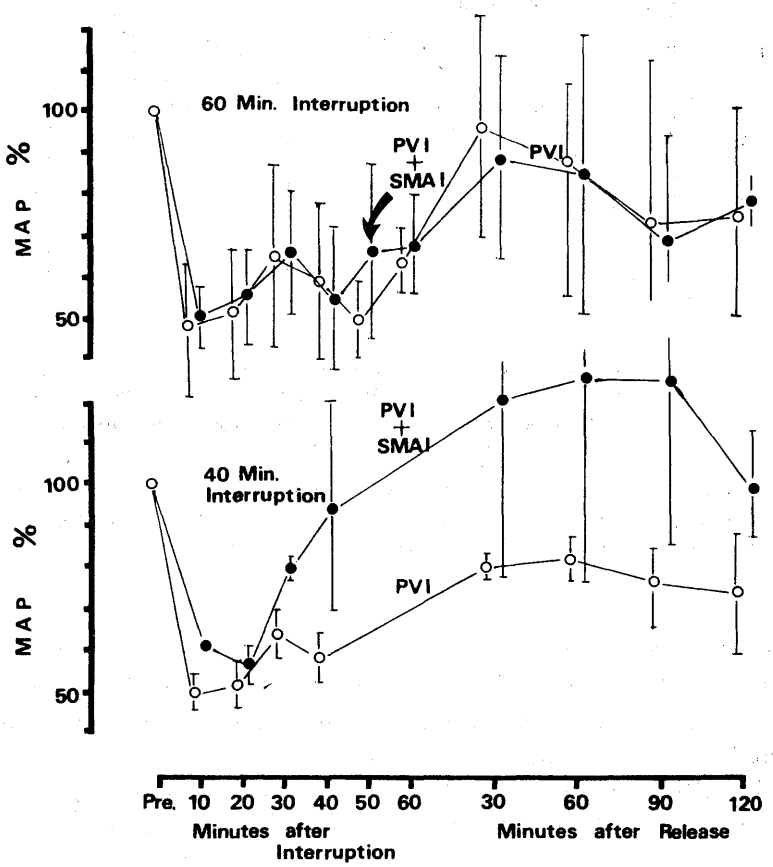

Fig. 1. Changes in Mean Arterial Pressure after Portal Interruption The mean arterial pressure levels in all groups fell to about 50% of the pre-interruption levels during the interruption, and did not return to the pre-interruption levels in other groups but Group IV two hours after release of the forceps.

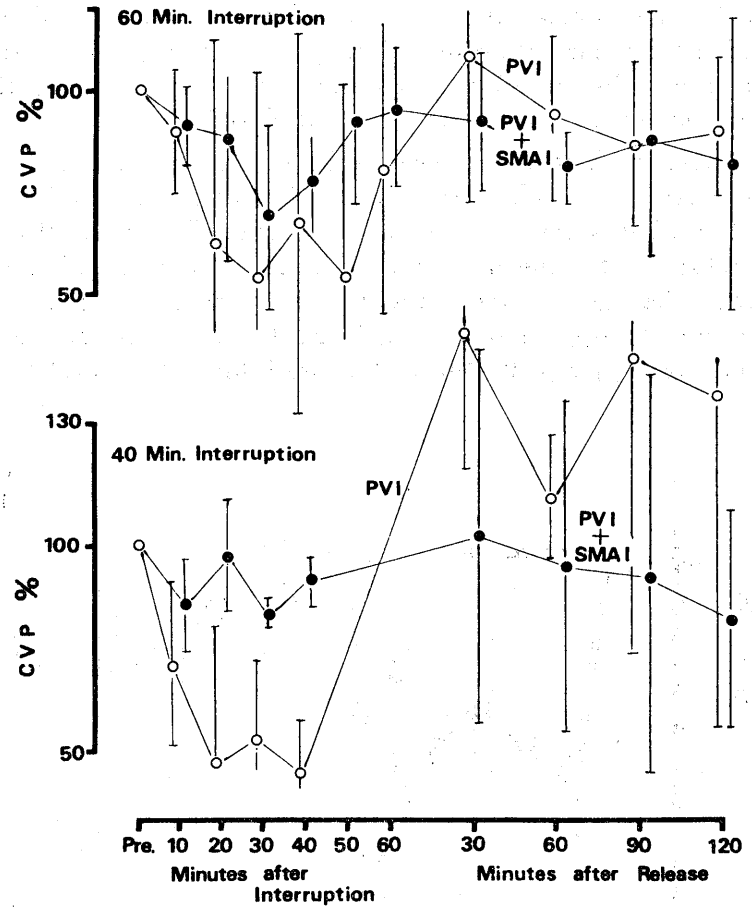

Fig. 2. Changes in Central Venous Pressure after Portal Interrup tion. Although the central venous pressure levels in all groups decreased during the interruption, these changes were less marked in Groups II and IV than in Groups I and III.

after release of the forceps and returned to normal later, reaching only 99.2 ± 13.9% of the pre-interruption levels in two hours (Fig. 1).

The central venous pressure levels in all groups decreased markedly immediately after interruption and recovered their pre-interruption level thirty min. after release of the forceps. However, these changes were less marked in Groups II and IV than in Groups I and III (Fig. 2).

The portal venous pressure levels in all groups increased remarkably immediately after interruption: namely, 443.6±157.9% of the preinterruption level in Group I, 356.4±74.9% in Group IV ten min. after interruption. These levels were maintained during the interruption and fell to

pre-interruption levels immediately after release of the forceps. Even though the portal venous pressure levels were much higher in Groups I and IV than in Groups II and III, there were no statistically significant differences. The levels in Groups I, II, III and IV two hours after release of the forceps were 102.3±33.8%, 105.8±8.2%, 106.8± 33.2% and 80.4±25.6% of the pre-interruption levels, respectively (Fig. 3).

All the dogs tolerated interruption of the portal vein for forty - and sixty - min. The dogs that received the portal vein interruption combined with the superior mesenteric arterial interruption were hemody-

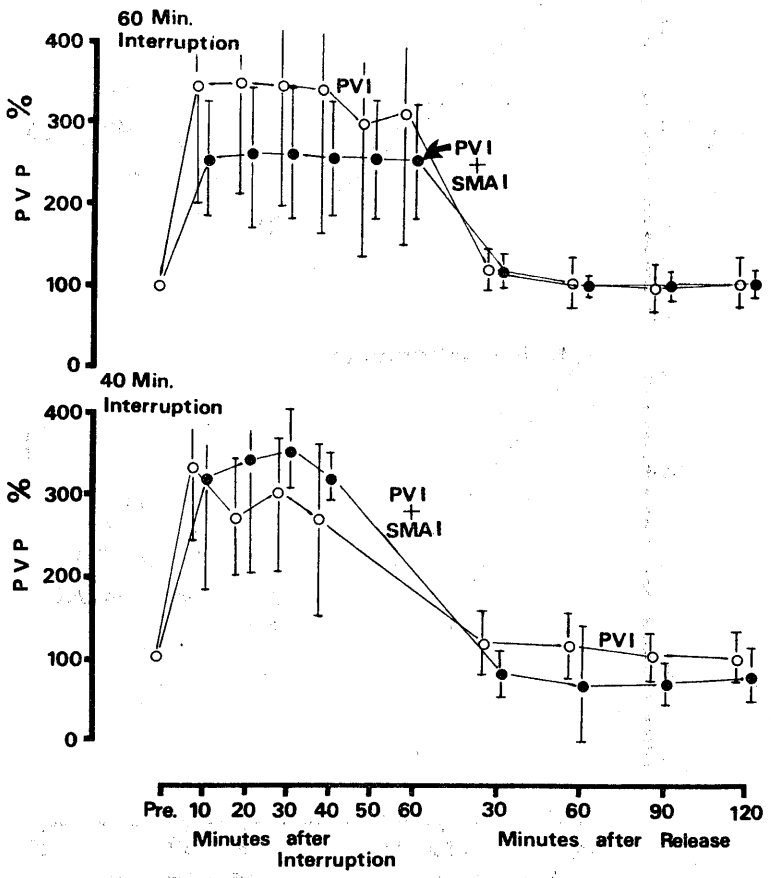

Fig. 3. Changes in Portal Venous Pressure after Portal Interruption. The portal venous pressure levels in all groups increased remarkably during the interruption and fell to pre-interruption levels immediately after release of the forceps. These changes during the interruption was more marked in Groups I and IV than in Groups II and III, though there were no statistically significant differences found.

namically more stable than the dogs that received the simple portal vein interruption.

Arterial pH levels in all groups decreased after the interruption: i.e., a decrease to 7.28±0.19 in Group I, 7.28±0.01 in Group II, 7.26±0.18 in Group III and 7.33±0.08 in Group IV. These levels, however, did not increase immediately after release of the forceps, but remained at their lowest levels for thirty min., then gradually rose. The levels two hours after release of the forceps were 7.294±0.16 in Group I, 7.281

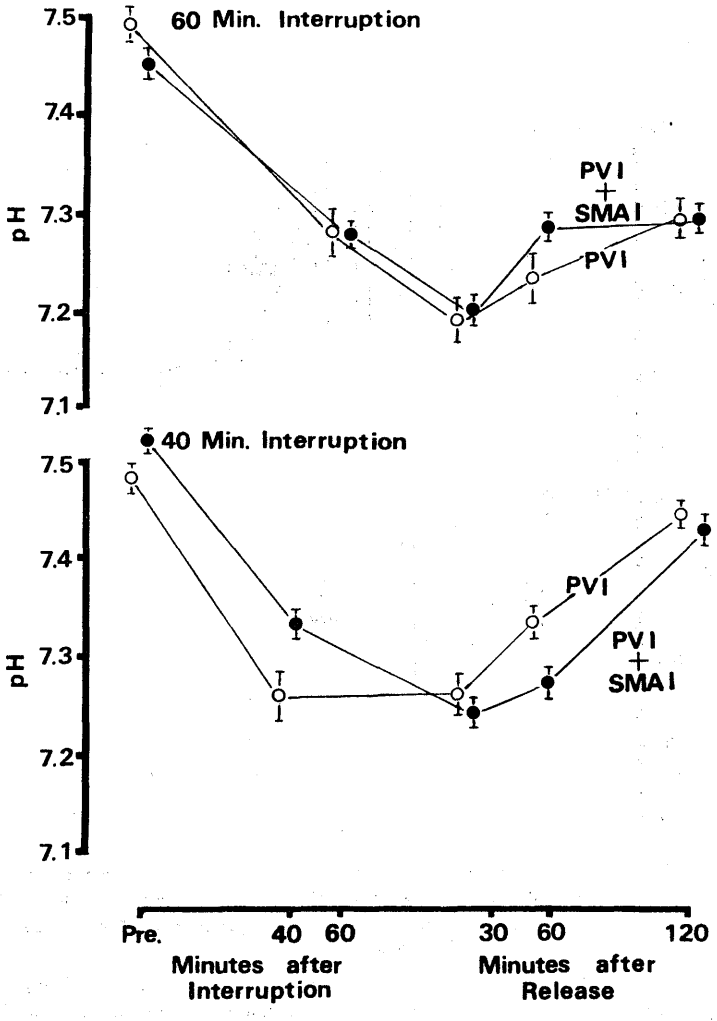

Fig. 4. Changes in Arterial pH after Portal Interruption Arterial pH levels in all groups decreased after the interruption and did not recover their pre-interruption level two hours after release of the forceps.

±0.02 in Group II, 7.438±0.05 in Group III and 7.41±0.01 in Group IV. The arterial blood became acidotic after interruption of the portal vein. The degree of acidosis was more remarkable in the sixty-minute interruption groups and their recovery was slower than in the forty-minute interruption groups, though no statistically significant differences were found (Fig. 4).

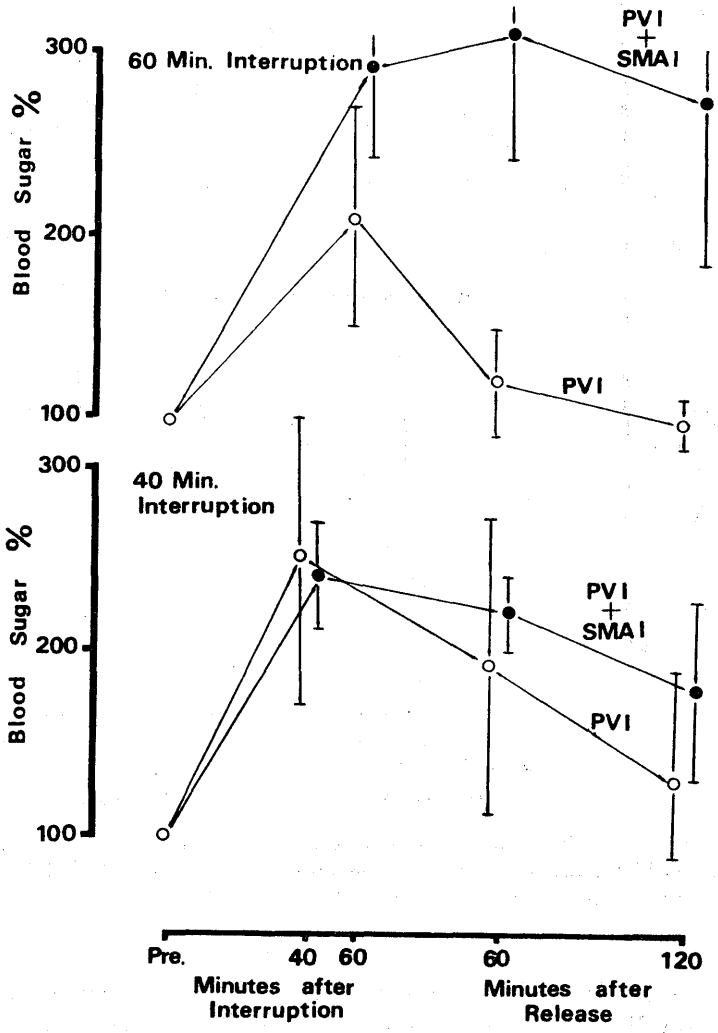

Fig. 5. Changes in Blood Sugar after Portal Interruption. Blood sugar levels increased remarkably during interruption and did not return after release of the forceps to the pre-interruption levels in all the groups except GroupI These changes was less marked in the simple portal interruption than in the interruption of the portal vein combined with interruption of the superior mesenteric artery.

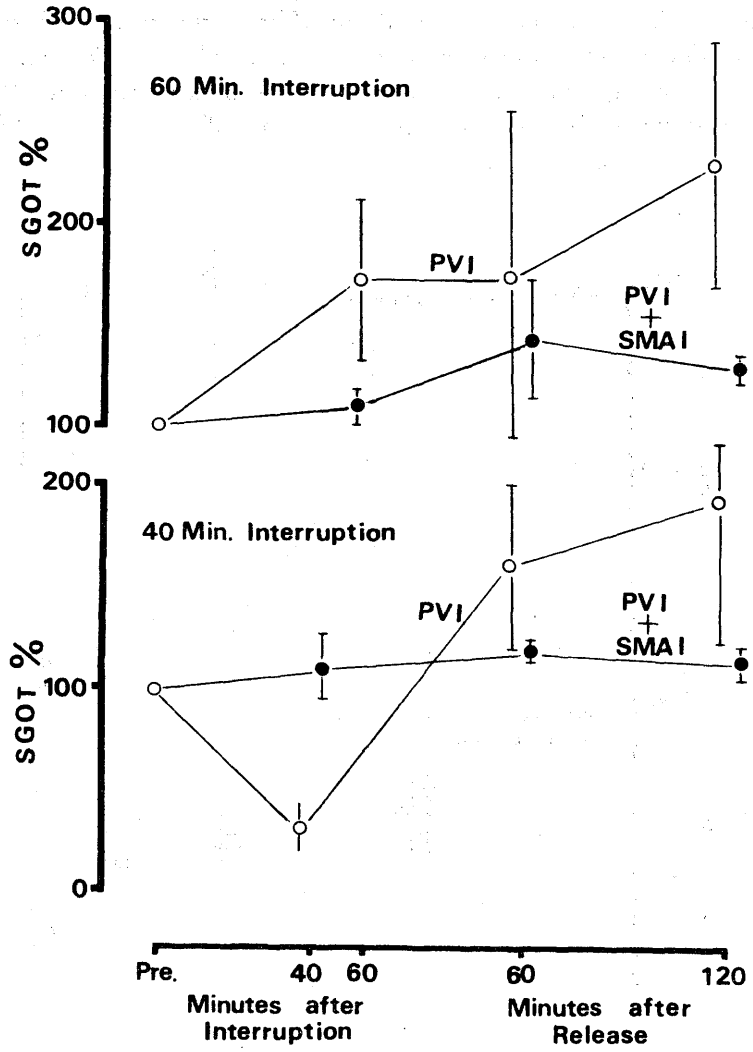

Fig. 6. Changes in SGOT after Portal Interruption. The SGOT in Groups I and III reached high levels after release of the forceps, but in Groups II and IV was almost unchanged during and after interruption

Blood sugar levels increased remarkably during interruption, especially in Group II. Except for Group II these levels decreased gradually after release of the forceps. In two hours the blood sugar levels were 98.1±12.4% of the pre-interruption level in Group I, 270 ± 91.1% in Group II, 127.9±69.8% in Group III and 179.2±54.5% in Group IV (Fig. 5).

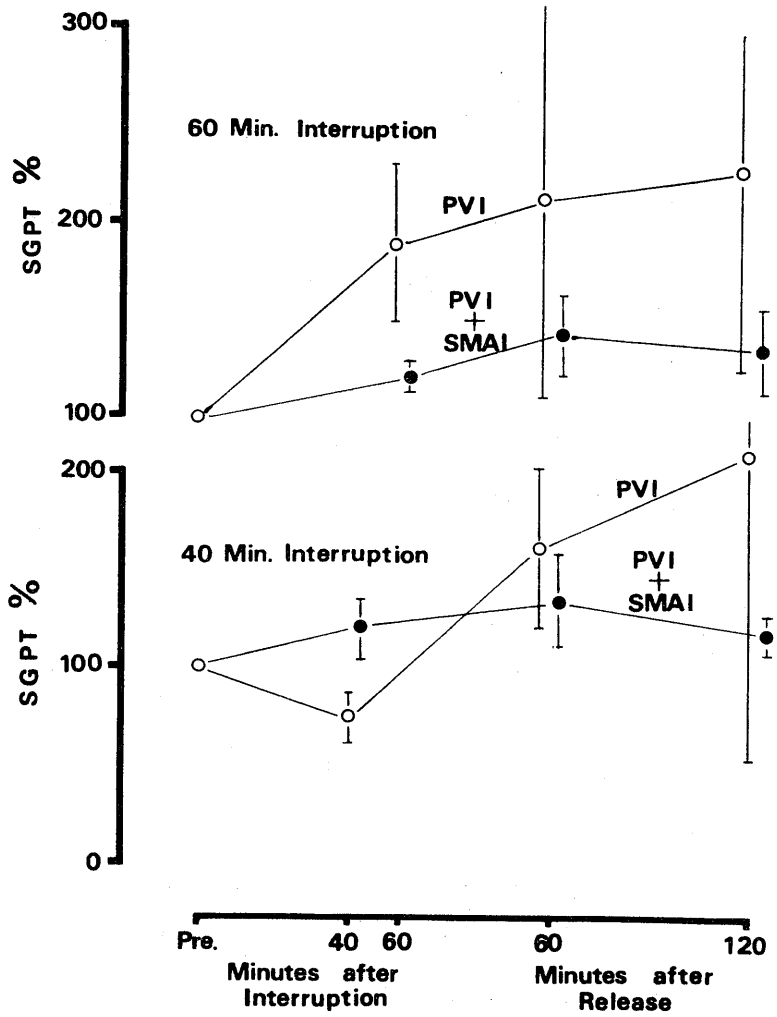

Fig. 7. Changes in SGPT after Portal Interruption. The SGPT in Groups I and III increased, but in Groups II and IV was almost unchanged after release of the forceps.

In the animals of Group I and III, the SGOT reached high levels after release of the forceps, when compared with the pre-interruption. Immediately before the release of the forceps the SGOT levels in Groups I and III were $172.1 \pm 48.6\%$ and $70.3 \pm 26.9\%$ of the pre-interruption levels, respectively. These levels increased gradually and two hours after release of the forceps reached $224.3 \pm 69.5\%$ of the pre-interruption levels in Group I and $196.0 \pm 75.0\%$ in Group III. On the other hand, in

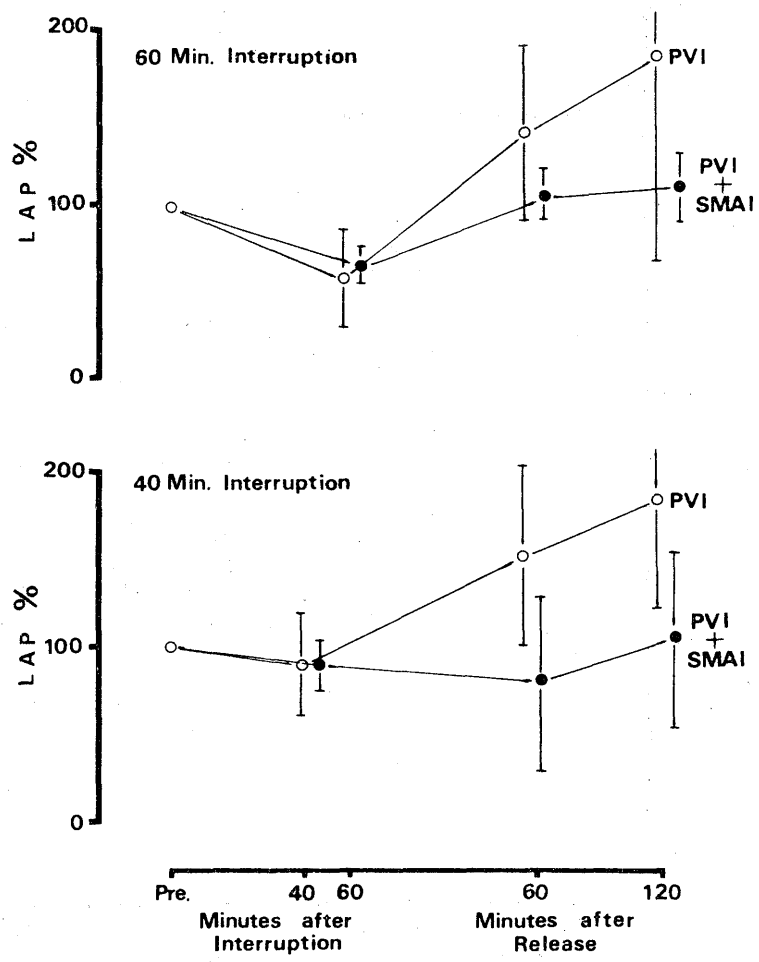

Fig. 8. Changes in Leucine Aminopeptidase after Portal Interruption. The leucine aminopeptidase levels in all groups fell briefly during the interruption. After release of the forceps, these levels in groups with the interruption of the portal vein combined with interruption of the superior mesenteric artery was not changed, while in groups with the simple interruption increased remarkably after release of the forceps.

Groups II and IV, the SGOT was almost unchanged during and after interruption regardless of the duration of interruption (Fig. 6).

Immediately before release of the forceps, the SGPT increased in Group I to $191.0 \pm 143.9\%$ and decreased in Group III to $74.1 \pm 11.8\%$ of the pre-interruption level, but these levels increased to $220.6 \pm 103.3\%$ of the pre-interruption level in Group I and $211.5 \pm 140.6\%$ in Group

III two hours after release of the forceps. However, the SGPT levels in Groups II and IV did not increase significantly during and after interruption: 129.6±25.3% of the pre-interruption level in Group II and 118.0±12.0% in Group IV two hours after release of the forceps (Fig. 7)

The leucine aminopeptidase levels fell slightly to 63.5±32.1% of the pre-interruption in Group I, 67.1±9.9% in Group II, 92.0±30.7% in Group III and 91.4±15.4% in Group IV just before release of the forceps. These levels in Groups I and III gradually increased to 185.6± 126.4% and 186.9±60.5% of the pre-interruption level, respectively, two hours after release of the forceps, but remained unchanged in Group II and IV (Fig. 8).

DISCUSSION

In recent years, the necessity of surgical procedures upon the portal vein has been emphasized due to the remarkable advances in liver and pancreas surgery. It is known that sudden occlusion of the portal vein in man is followed by death within a matter of hours. The safe period for acute interruption of the portal vein under normothermic conditions has been reported to be about 1 hour by Elman and Cole[3] and 50 to 90 min. by Neuhof[4]. However, Oyanagi[5] reported that a permissible safe period was only thirty minutes, based on his experimental results using dogs. In our results, a safe period was not determined, because the animals were observed for only two hours after cessation of interruption. A number of papers have been published concerning the cause of death after acute portal venous interruption, such as intoxication, neurogenic origin and hemorrhagic shock. Mays[1] attributed the cause to the accumulation of all the blood into the vessels of the intestines and consequent exsanguination of the brain and other organs. Schiff[6] postulated that it was due to toxemia. Elman et al.[3] reported that rapid death was based on the loss of blood from the systemic into the portal circulation of a sufficient magnitude to reduce blood pressure. Johnstone[7] found that thirty min. following acute ligation, the circulating blood volume had decreased by 57.9% but stated that the cause of death could not be attributed solely to this decrease. In our present experiment, the portal venous pressure rose abruptly and the systemic arterial and central venous pressures dropped gradually as the portal venous interruption was carried on. The degree of change was smaller when the portal venous interruption was combined with a superior mesenteric interruption than when only a simple portal venous interruption was performed. Consequently, cause of death after acute portal interruption could be due

to a decrease in the circulating blood volume caused by congestion in the splanchnic area.

The arterial blood pH fell during interruption of the portal vein and its restoration did not occur within two hours after cessation of the interruption. Backlund et al.[8] reported that pH levels of 7.02 or less were recorded at the end of two hours following cessation of interruption because of metabolic acidosis.

Blood sugar was elevated during interruption of the portal vein and its degree was more severe in the portal vein interruption combined with the superior mesenteric arterial interruption than in the simple portal venous interruption. Though its cause was not clearly determined, the function of the pancreas may have been reduced by the diminution of pancreatic blood flow.

There are only a few reports on the effects of portal venous interruption on SGOT, SGPT and leucine aminopeptidase[5]. Oyanagi found that the SGOT reached a high level immediately after the cessation of a 30-minute interruption of the portal vein. This level was maintained for 24 to 48 hours because of extended subendocardial ischemia, probably due to decrease in circulating blood volume occurring necessarily after interruption of the portal vein. In our results, SGOT, SGPT and leucine aminopeptidase rose 1 to 2 times higher than normal two hours after cessation of interruption probably due to hepatic ischemia. The degrees of change were less in the portal vein interruption combined with a superior mesenteric arterial interruption than in the simple portal vein interruption.

It seems safer to interrupt the portal vein along with the superior mesenteric artery than to interrupt only the portal vein, though human reaction to sudden portal vein interruption differs from the reaction of rabbits, cats and dogs[9].

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