TABLE I.—**Clinical Findings**

Case no.	Sex, age, indications for vascular surgery	Arteriographic findings	Operation	Mechanism of acute ischemia	Localization of ischemia and diagnostic delay	Medical complications	Surgical treatment	Outcome
1	Male, 76 years elective infrarenal aorta aneurysmectomy		Aortoiliac prosthetic graft	Clamping	Right and left colon D12	ARDS hemodialysis digestive hemorrhage	+ D15 total colectomy cholecystectomy	Death + D70 multiple organ failure persistent peritonitis
2	Male, 58 years elective infrarenal aorta aneurysmectomy	Celiac axis SMA, IMA normal	Aortobiiliac prosthetic reimplant IMA	Thrombocytopenia due to heparin	Small intestine Right and left colon D2	Moderate tubular disease	+ D6 thrombectomy graft and SMA	Favorable
3	Male, 64 years elective infrarenal aorta aneurysmectomy	Stenosis celiac axis SMA normal IMA occluded	Thrombo-endarterectomy aortic tube	Cholesterol embolism	Small intestine right and left colon liver, gallbladder D2	ARDS moderate tubular disease severe cytolysis	+ D2 exploratory laparotomy + D3 second look	Death + D3 multiple organ failure
4	Female, 74 years ruptured infrarenal aorta aneurysm	Stenosis celiac axis SMA normal IMA occluded	Aortic tube	Hemorrhagic shock	Left colon gallbladder D5	ARDS hemodialysis severe cytolysis	+ D5 Hartmann + D13 cholecystectomy peritonitis	Death + D62 infective pulmonary pathology
5	Male, 65 years ruptured, infected aortic		Aortobiiliac prosthetic graft	Hemorrhagic shock	Left colon D0		+ D1 Hartmann cholecystectomy	Favorable
6	Male, 60 years aortoiliac atherosclerosis	Celiac axis normal stenosis SMA IMA normal	Aortobiiliac prosthetic graft	Ligation IMA	Left colon D3		+ D28 left colectomy for cicatricial colonic stricture	Favorable
7	Male, 64 years aortoiliac atherosclerosis	Iliac axis normal SMA normal IMA occluded	Aortobifemoral prosthetic graft	Clamping	Right and left colon rectum D6	moderate cytolysis	+ D7 total coloprotectomy cholecystectomy + D9 closure + D20 fistule rectal stump	Favorable
8	Male, 79 years fissurated infrarenal aorta aneurysm		Aortic tube	Clamping	Left colon D8		Coloscopic surveillance	Favorable

ARDS = adult respiratory syndrome; SMA = superior mesenteric artery; IMA = inferior mesenteric artery

TABLE II.—**Clinical Findings**

Case no.	Sex, age, indications for vascular surgery	Arteriographic findings	Operation	Mechanism of acute ischemia	Localization of ischemia and diagnostic delay	Medical Complications	Surgical treatment	Outcome
9	Male, 67 years elective infrarenal aortic aneurysmectomy	Celiac axis normal SMA normal IMA occluded	Aortoiliac prosthetic graft	Clamping	Right and left colon D2		+ D2 Total colectomy + D4 recto operation for ischemia of ileostomy	Favorable
10	Male, 75 years elective infrarenal aortic aneurysmectomy	Celiac axis normal SMA normal IMA occluded	Thrombo-endarterectomy of interrenal aorta, aortobiiliac prosthetic graft implantation IMA	Clamping	Left colon D4		Coloscopic surveillance	Favorable
11	Male, 63 years elective infrarenal aortic aneurysmectomy	Stenosis celiac axis SMA normal IMA occluded	Aortic tube aorto left renal, PTFE graft	Cholesterol embolism	Left colon D2	Hemodialysis severe cytolysis intestinal hemorrhage	+ D2 Hartmann cholecystectomy	Death + D2 Necrotizing pancreatitis
12	Male, 60 years aortoiliac atherosclerosis	Celiac axis normal SMA normal IMA occluded	Aortobifemoral prosthetic graft	Clamping	Left colon D7		+ D8 Hartmann	Favorable
13	Male, 71 years elective infrarenal aortic aneurysmectomy	Celiac axis normal SMA normal stenosis IMA	Aortobiiliac prosthetic graft	Ligation IMA	Left colon D0	Moderate cytolysis	Coloscopic surveillance	Favorable
14	Male, 82 years ruptured infrarenal aortic aneurysmectomy		Aortobifemoral prosthetic graft	Hemorrhagic shock	Left colon D0	Hemodialysis moderate cytolysis	+ D6 Hartmann	Death + D14 left and right colonic necrosis
15	Male, 82 years ruptured infrarenal aortic aneurysm	Celiac axis normal SMA normal IMA normal	Aortobifemoral prosthetic graft	Clamping	Left colon and rectum D3	ARDS moderate tubular disease moderate cytolysis digestive tract hemorrhage	+ D3 Hartmann + D4 Abdomino-perineal resection cholecystectomy	Favorable
16	Male, 66 years elective infrarenal aortic aneurysmectomy	IMA occluded	Aortic tube	Clamping	Left colon D6	Moderate tubular disease moderate cytolysis	+ D6 Hartmann + cholecystectomy + D10 small intestinal fistula	Favorable

ARDS = adult respiratory syndrome; SMA = superior mesenteric artery; IMA = inferior mesenteric artery

TABLE III.—**Clinical signs of acute colorectal ischemia**

	Patients	Percent
Temperature > 38°5 < 36°5	11	69%
Diarrhea	10	62%
Shock	9	56%
Left iliac fossa	7	44%
Intestinal obstruction	6	38%
Rectal bleeding	5	31%
Bacteremia	2	12%

gilis associated with *Clostridium perfringens* in one, and *Escherichia coli* and *Clostridium perfringens* in the other. Coloscopy was performed in eight patients (50%) and in all cases led to the suggestion of the correct diagnosis. Biopsies were obtained in six of these patients and always confirmed the diagnosis. Blood was drawn for analysis between the evening before and the third day after diagnosis. Results of biologic abnormalities are reported in Table IV.

Thirteen patients (80%) underwent reoperations. The ischemic colon was removed in 11 (70%). In one patient, extended intraabdominal necrosis (liver, spleen, pancreas gallbladder, small and large intestines) without therapeutic potential was found. A second look was performed in six patients, and in four a complementary surgical ablative procedure was necessary. Of seven cholecystectomies, three were performed for acute cholecystitis. In Patient 2, complete restoration of the colonic vascularity was obtained after thrombectomy.

In cases of adult respiratory distress syndrome, the mean duration of mechanical ventilation was 43 days, compared to 12 days without. Overall mortality was 31% (5 of 16) with an average follow-up of 11 months. Two patients who died were in the group operated on for infrarenal aortic aneurysm. In five cases death was directly related to acute colorectal ischemia, persisting ischemia of the small or large intestine in two cases, and multiple organ failure related to sepsis other than acute colorectal ischemia in three cases (pneumonia, persistent peritonitis in spite of several iterative laparotomies, and pancreatitis with necrosis).

The mean age of patients who died was 71.8 years compared with 64.5 years for survivors (NS). The best predictive factors for mortality (Table V) were kidney tubular disease and severe hepatic cytolysis. The SSI was calculated on the day the diagnosis of acute colorectal ischemia was made, usually the day of admission to intensive care. The SSI score was 18 on an average in the group of patients who died (range 16 to 22) and 11 in survivors (range 6–15) ($p < 0.01$).

DISCUSSION

The incidence of acute colorectal ischemia in our series of aortic surgery was 2.8%. This can be broken down to 3.4% for surgery of infrarenal aortic aneurysms and 1.9% for aortoiliac surgery, which is comparable to the literature. Ernst and colleagues [4], employing routine colonoscopic examination, found an incidence of 7.4% and 4.3%, respectively. Even though these two studies are not comparable, the true frequency of acute colorectal ischemia could be underestimated by 50 to 60% in the absence of routine colonoscopy. Vascular visualization permits delineation of the type of colonic vascularity most threatened by aortic clamping, as is essentially the case when the inferior mesenteric artery is occluded. According to our study, two trigger mechanisms of acute colorectal ischemia can be identified: When it is necessary to clamp the infrarenal aorta, certain patients experience transient ischemia of the colon. In this case, colonic vascularization is restored quickly and remains identical to preoperative status.

In other patients, ischemia is prolonged, either because of perioperative hemorrhagic shock or ligation of a patent inferior mesenteric artery with previous transverse colectomy (a mechanism already described [13]) or prosthetic thrombosis due to severe thrombocytopenia secondary to heparin, or cholesterol embolism. No one mechanism can be incriminated in the onset of acute colorectal ischemia. Experimental studies have shown that ischemia of short duration can lead to irreversible necrosis. Conversely, in the case of initial ischemia, cellular anoxia is not the only cause of tissular lesions.

The role of oxygen-derived free radicals has recently been demonstrated [14,15]. These molecules are highly reactive and are generally produced in small quantities by enzymes such as xanthine oxidase in the small intestine or aldehyde oxidase in the colon [16]. The intestinal cells possess several enzymatic systems capable of eliminating these free radicals, including superoxide dismutase [10,17,20]. During intestinal ischemia, enzymes capable of synthesizing free radicals derived from oxygen and their substrata are produced in large quantities. Reperfusion, i.e. oxygen delivery, is responsible for massive production of free radicals. These mole-

TABLE IV.—**Biological changes**

	Less than normal	Normal	More than normal
Serum phosphorus	43%		57%
Alkaline phosphatases	0	66%	34%
Aspartate aminotransferase	0	30%	70%
Alamine aminotransferase	0	67%	33%
Lactic dehydrogenase	0	15%	85%
Creatine phosphokinase	0	38%	62%

TABLE V.—**Prognostic criteria of mortality**

	Deaths	Survival	CHT
Iterative intestinal ischemia	40	27.3	NS
Transmural necrosis	80	63.7	NS
Emergency vascular surgery	40	18.2	NS
Visceral failures	—	—	—
Digestive tract hemorrhage	40	0	<0.05
ARDS	60	0	<0.05
Tubular disease clearance < 5 ml/minute	60	0	<0.01
Cytolysis > 3 × N	60	9.1	NS
Shock	100	27.3	<0.05
More than 2 visceral failures	100	9.1	<0.01
Simplified severity index	18	11	<0.01
Age	71.8	64.8	NS

ARDS = adult respiratory distress syndrome

cules possess major cellular toxicity and are responsible for tissular lesions. It has been shown experimentally that dismutase superoxide and inhibitors of xanthine oxidase reduce the permeability of the intestinal wall and attenuate the intensity of histological lesions, the risk of necrosis, of perforation, and mortality [11,14,15,21–23].

Aminophylline, a substrate of xanthine oxidase, increases histological lesions [14]. In experimental models of severe and prolonged ischemia, the introduction of superoxide dismutase did not modify the permeability of the intestinal wall or the intensity of histological lesions [11]. Under these circumstances, free radicals are not a determinant in the onset of intestinal necrosis: tissular lesions are induced by arterial ligation, and in the absence of sufficient flow, necrosis ensues. This model corresponds best to the acute colorectal ischemia seen in humans after alteration of colonic vascularity, prolonged low-flow states, or cholesterol embolism, and is best named "devascularization acute colorectal ischemia". Conversely, during isolated clamping of pathological arteries inducing less severe ischemia, the action of free radicals could be responsible for the observed lesions. This model could explain why acute colorectal ischemia can occur in the absence of altered colonic vascularity and is named "ischemia reperfusion acute colorectal ischemia." Clear demonstration of the role of free radicals in humans is a prerequisite for evaluation of the therapeutic options for colonic protection which have been experimented in the animal.

Arteriographic evaluation of patients at risk could help decrease the incidence of acute colorectal ischemia. When performing aortic reconstruction, it seems preferable to perform inferior mesenteric artery revascularization irrespective of the results of residual pressure measured during operation. In the absence of revascularization, postoperative hypotension can be responsible for colonic ischemia. This is the only well-established preventive surgical procedure for acute colorectal ischemia [24].

Colonoscopy is the primary investigational tool [25], as it is diagnostic and permits the procurement of specimens for histopathological confirmation. Routine use of colonoscopy could lead to earlier diagnosis. As colonoscopy is simple and not dangerous, this investigative method should be entertained routinely whenever aortic surgery is followed by diarrhea, cerebral disorders, or cardiac, respiratory, or renal failure, even when these entities occur in an isolated fashion. The reported severity of acute colorectal ischemia is most likely due to the delay or absence of proper diagnosis.

Several studies, in animals as in humans, have attempted to find sensitive and specific biological markers for colonic ischemia: In animals these are serum titers of phosphorus [26–28], urea, uric acid [27], LDH [27], CPR [27,29–32], ASAT and ALAT, alkaline phosphatases [27], hexosaminidase [33], vasoactive intestinal peptide (VIP) [34–36]. In humans, CPK [37–39], LDH [39], hexosaminidase [40] and phosphorus [28,41] have been found to be abnormally elevated. It is not known, however, whether or not these markers are specific. Results were difficult to interpret in our 16 patients. Variation of serum phosphorus is not unusual after major surgery. Heavy glucose infusion and alkalosis due to mechanical ventilation can be responsible for the hypophosphoremia observed after aortic surgery. On the other hand, hyperphosphoremia is usually seen only when kidney failure occurs. In two of our patients, however, hyperphosphoremia was seen with normal kidney function, and no cause could be found.

As often occurs after surgery, CPR and LDH were often found to be elevated. High titers of alkaline phosphatases, ASAT and ALAT, when present, are most likely indicative of hepatic failure associated with acute colorectal ischemia [42]. While these abnormalities may often constitute alarm signals, they do not contribute to the topographical diagnosis of postoperative colonic complications. Because of their pharmacological properties, elevated serum levels of hormones usually found in the colon wall could be a partial explanation for the clinical signs observed [43,44]. A prospective study of the dosage of colonic enzymes and hormones is currently underway.

Surgery is indicated according to the aspect of the colonic mucosa and associated signs of severity. When superficial ischemia is found on colonoscopy, operation is mandatory even when only one sign generally associated with infection, such as cerebral disorders, septic shock, pulmonary edema, acute tubular disease, hepatic cytolysis, or acute gastroduodenal ulceration, is present. The surgical procedure performed depends on the condition of the colon. Ideally all necrotic colon must be resected. In our opinion, the frequency with

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