CORONARY DISEASE AMONG UNITED STATES SOLDIERS KILLED IN ACTION IN KOREA

PRELIMINARY REPORT

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The purpose of this paper is to describe and analyze the gross lesions found in the coronary arteries of United States soldiers killed in action in Korea. The histology will be discussed in detail in a subsequent paper as will such pertinent data as race, body build, and personal habits.

MATERIAL

Recently 300 autopsies were performed on United States battle casualties in Korea. Most of these soldiers were killed in action or suffered accidental death in front line areas. The coronary arteries were carefully dissected in all cases. No case in which there was known clinical evidence of coronary disease was included in this series.

The average age in 200 cases was 22.1 years. The ages in the first 98 cases were not recorded except that the oldest patient was 33. In the entire series, the youngest recorded age was 18 and the oldest 48.

FINDINGS

In 77.3% of the hearts, some gross evidence of coronary arteriosclerosis was found. The disease process varied from "fibrous" thickening to large atheromatous plaques causing complete occlusion of one or more of the major vessels (table 1).

Occlusion was considered complete only when the plaque had no free surface and was fused to the wall opposite its point of origin. A conservative attitude in estimating the amount of luminal narrowing was maintained throughout. No actual measurements were made.

TABLE 1.—*Percentage of Cases Showing Varying Amounts of Luminal Narrowing*

Amount of Luminal Narrowing	% of Cases
"Fibrous" thickening or streaking causing insignificant luminal narrowing	35.0
Plaques causing luminal narrowing over	
10%	23.3
20%	6.3
30%	3.7
40%	3.0
50%	3.0
60%	1.7
70%	1.0
80%	1.3
90%	5.3
Plaques causing complete occlusion of one or more vessels	3.0

In several of the cases listed in table 1 as showing over 90% luminal narrowing, the plaques completely filled the lumen but displayed a free surface not fused with the intima. Hypertrophy was not observed in any heart examined. The size could not be correlated with the severity of disease found in the coronary arteries.

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In the great majority of cases, the location of the lesions followed a constant pattern. If the lesion was found in the proximal third of the left coronary artery, it was usually thickest on the epicardial side of the lumen, whereas, if it was in the distal third of the artery just proximal to the bifurcation of the circumflex artery, it tended to assume a more medial position. At the bifurcation, the plaque was constantly found on the wall of the left coronary artery directly opposite the ostium of the circumflex artery (fig. 1). In the upper fourth of the anterior descending artery, the disease process was usually found on the medial posterior wall of the vessel. More distally, the posterior or myocardial side of the lumen was the common location (fig. 2*A*). The medial branch of a bifurcating anterior descending artery was always more severely involved in cases showing extensive disease (fig. 2*B*). In some cases, plaques were encountered on the epicardial side of the lumen of the anterior descending artery; these plaques were opposite the bifurcation of a septal branch.

TABLE 2.—*Sites of Lesions Found in Coronary Arteries at Autopsy*

Site	No. of Cases
Left coronary	
Above bifurcation	10
At bifurcation	22
And circumflex	1
And right coronary	1
Circumflex, and right coronary	1
Anterior descending	
Branch	114
And circumflex	13
And right coronary	38
And left coronary	5
Left and right coronaries	4
Left coronary, and circumflex	1
Circumflex, and right coronary	8
Left and right coronary, and circumflex	11
Right coronary only	3
Circumflex only	4

The pattern of disease was not as constant in the circumflex artery as it was in the left and anterior descending branches. Streaking and/or plaque formation was oftenest observed on the superior and posterior walls of the proximal fourth of the vessel, while the next most frequent site of involvement was the posterior wall in the middle third of the vessel, usually opposite a bifurcation of the circumflex artery.

In the proximal fourth of the right coronary artery, small plaques were commonly encountered on the posterior wall opposite the adipose branches. Elsewhere in the right coronary, plaques were usually found on the posterior and superior walls. This was especially true at the right border of the heart opposite the origin of the right marginal artery.

Disease was most commonly found at the bifurcation of the circumflex artery and in the 3 cm. of anterior descending artery distal to the bifurcation (table 2). In 54 of the 112 cases with lesions in the anterior descending artery, the disease process started at the bifurcation of the circumflex artery and extended down the anterior descending branch, usually on the medial and posterior sides of the lumen. In every instance in which the diseased anterior descending artery bifurcated in the first 2 cm., the deposition of atheromatous material appeared to be intensified. The angle formed by the anterior descending and circumflex arteries is important. An angle approaching 80 degrees always seemed to predispose the artery to the development of a plaque. On the other hand, the disease process tended to be modified when the circumflex artery arose at the ostium of the right coronary artery, the vessel coursing around the base of the aortic and pulmonary arteries to assume its usual position in the atrioventricular septum on the anterior surface of the heart. Minimal disease was found in the left coronary artery in three such cases.

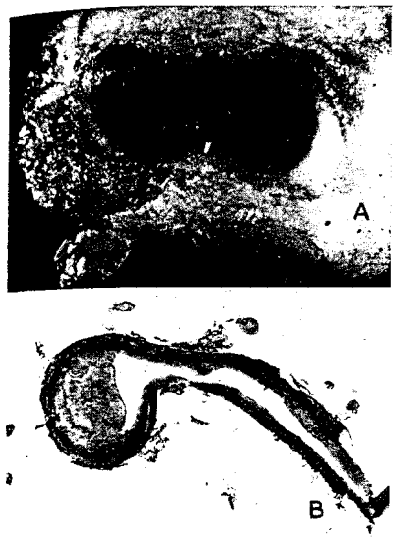

Fig. 1.—*A*, plaque formation in left coronary artery opposite ostium of circumflex (× 4). *B*, detail of atheromatous plaque shown in *A* (× 14).

COMMENT

It is apparent that the lesions are commonly located at or near points of bifurcation; this gives the impression that hemodynamics in the lumen about bifurcations must play an important part in the mechanism of plaque formation.

The phasic aspect of the coronary circulation is mainly the result of the aortic blood pressure and the contractions of the myocardium during the cardiac cycle. Gregg and Green [1] have shown experimentally that, during isometric contractions and ejection, the blood in the deeper and more strongly compressed vessels in the myocardium is forced backward into the larger proximal channels. Wood [2] stated that, during systole, ventricular contractions prevent blood from flowing through the coronary vessels that penetrate the left ventricular muscles, while the larger arteries on the surface dilate to form an elastic reservoir that recoils during diastole and acts as an accessory pump forcing the blood onward. During ventricular relaxation, the blood is able to flow through the vessels penetrating muscle, because it is propelled by the aortic diastolic pressure and by the recoil of the elastic reservoir. The compression of the intramural arteries is, as noted by Friedberg,[3] less in the right ventricle.

We maintain that eddying about bifurcations of the coronary arteries, occurring during the diastolic recoil of the dilated coronary arteries, is a dynamic factor in the mechanism of plaque formation. As has been shown, the most frequent sites of plaque formation are on the medial wall of the left coronary artery at the bifurcation of the circumflex artery and on the medial and posterior sides of the proximal fourth of the anterior descending artery. The severest disease was found in those cases in which the bifurcation of the circumflex artery formed an 80 degree angle and the anterior descending artery gave rise to a large branch just distal to the origin of the circumflex artery. It is postulated that under such conditions

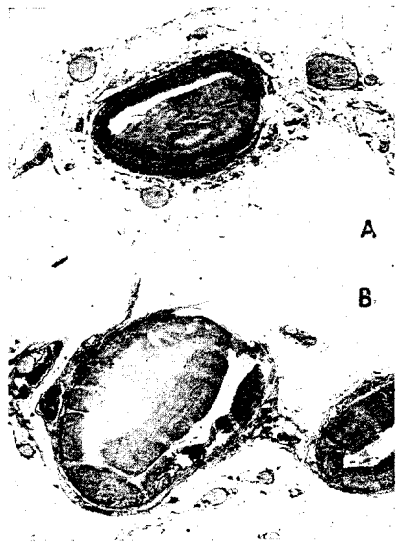

Fig. 2.—*A*, large plaque on myocardial side of anterior descending branch (× 12.5). *B*, large plaque in medial branch of a bifurcating anterior descending artery (× 18.5).

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eddying of blood in the left and anterior descending arteries traumatizes the intima and, thus, starts the vicious circle of plaque formation.

Many factors have been mentioned as the primary cause of coronary arteriosclerosis. Heredity has been accepted by many as an important agent in the development of coronary disease. This may be manifested in the inherited pattern of coronary artery distribution. It has long been realized that coronary artery disease is much commoner in young men than in young women [4]; this suggests a hormonal factor. Boas and Ludwig [5] have shown experimentally that testosterone stimulates the formation of connective tissue, while estrogens inhibit connective tissue formation. Some investigators have emphasized the ratio of serum cholesterol to phospholipids. According to Gofman and associates,[6] at any total serum cholesterol level, the S_f 12-20 and S_f 20-100 classes of lipoproteins are higher in patients with coro-

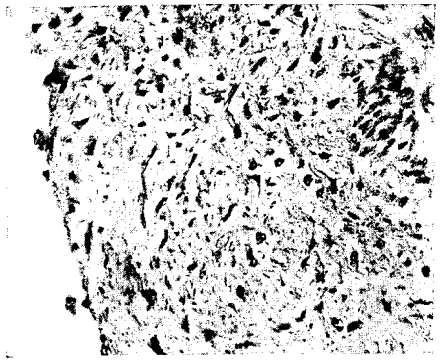

Fig. 3.—Thick intima in artery of a young Korean prisoner of war (× 33.5).

nary disease than in normal subjects.[7] Winternitz, Thomas, and Le Compte [8] demonstrated with histological preparations and carefully executed reconstructions that intramural hemorrhage may furnish the bulk of cholesterol material in arteriosclerotic lesions, while Wartman and Laipply [9] produced intramural hematomas experimentally and caused sclerosis in 30% of their cases. Saphir and Gore [10] presented evidence of an inflammatory basis of arteriosclerosis.

Of special interest is the work of Dock [11] in which he demonstrated that the coronary intima of the male newborn is thicker than the intima of the female newborn. Zink [12] and Lack [13] maintain that this phenomenon occurred only at points of bifurcation.

Most investigators are of the opinion that these areas of thickening are developmental irregularities representing prearteriosclerotic or early arteriosclerotic lesions.

It is interesting to note that the intima of the coronary vessels in young Koreans is thick (fig. 3), although coronary atheromatosis is said to be rare in that race. Here we have an anatomic feature indicating stress, but plaque formation is rare. This would strongly suggest dietary factors. It is most unlikely that one factor alone can be indicated as the cause of coronary sclerosis. The findings presented in this paper indicate wear and tear on the intima caused by eddying of blood at points of bifuration. However, other basic factors, such as hormones and diet, must be present before the disease progresses to plaque formation.

Two important points should be emphasized in view of observations made in this study. 1. Thrombosis occurs more commonly in diseased arteries, especially in cases in which extensive trauma and shock exerted their influence (fig. 4). 2. In cases of sudden or clinically unexplained death of young men, the presence of a completely occluded coronary artery is not necessarily the cause of death.

Fig. 4.—Thrombosis of anterior descending artery in a 21-year-old white man who died three days after severe injuries (× 21.5).

SUMMARY

The coronary arteries of 300 soldiers killed in action were dissected. The average age of the men in this group was approximately 22.1 years. In 77.3% of the cases, gross evidence of coronary disease was demonstrated that varied from "fibrous" thickening to complete occlusion of one or more of the main branches. The exact locations of the lesions were shown to be at or near points of bifurcation of the vessels. The hemodynamics of the coronary circulation as modified by the anatomic variations of the coronary arterial tree are implicated as an important factor in the formation of plaques.

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PRIMARY RESECTION OF THE COLON IN ACUTE ULCERATIVE COLITIS

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Acute fulminating ulcerative colitis is a serious disease that carries a very high mortality. The acute attack may be an exacerbation of preexisting disease or it may represent the first manifestation of colitis in a previously apparently healthy person. In either case, the patient is acutely ill with high fever, diarrhea, bloody and purulent stools, and an overwhelming toxemia. There is often evidence of peritoneal irritation or frank peritonitis. Because these patients are so acutely ill, there has been a tendency to avoid surgical treatment, and, when it has become inevitable, simple ileostomy has been accepted as the operation of choice.[1] The results of this therapy have been poor, and the mortality has ranged from 20 to 60% in reported series of cases. In analyzing the deaths that have occurred in the past, certain facts become apparent: 1. The majority of patients show evidence of severe blood and protein loss, and this continues even in the presence of a functioning ileostomy. The blood loss may amount to 200 to 300 cc. and the protein loss to 150 to 200 gm. in 24 hours. The source of this loss is the diseased colon, and replacement in a severely ill patient is difficult and at times impossible. Ileostomy adds the factor of fluid and electrolyte loss. 2. Infection is an important factor in mortality. Sepsis and toxic absorption are universally present, and perforation of the colon is common (it occurred in 20% of fatal cases). The source of infection is primarily the diseased colon (fig. 1).

Since all of these factors are derived from the diseased bowel, the logical solution would be its removal, and, since primary resection of the colon is so well tolerated in debilitated patients with chronic ulcerative colitis,[2] it has appeared worth while to evaluate the use of this procedure in acute fulminating cases of the disease. My experience with primary colectomy now covers 120 cases of ulcerative colitis, and in this group there have been five operative deaths. Forty-three of these cases fall into the acute category, and it is this group I wish to consider in detail.

INDICATIONS FOR SURGERY

Since acute ulcerative colitis responds to medical therapy in many cases, it is obvious that any radical surgical procedure should be reserved for cases in which it fails. This fact has led to the attitude that surgery is indicated only as a last desperate measure, and patients are referred for surgery in moribund condition. In an attempt to avoid this, I have adopted the following indications for surgical treatment. 1. The first is failure of medical therapy to produce a remission within a three week period. This period is arbitrarily chosen, but it has been my experience that if a patient does not begin to improve within three weeks, further delay is dangerous. During this interval, patients have been treated with transfusions, antibiotics, corticotropin (ACTH), methantheline (Banthine), and, in some instances, psychotherapy. Sixty per cent of the patients have responded to this treatment, and the other 40% have undergone surgical treatment. 2. The second indication is perforation, actual or impending. In the presence of frank peritonitis or free gas in the peritoneal cavity, surgical intervention becomes an emergency matter. In any patient in whom localized abdominal tenderness develops, impending perforation is suspected, and in these instances a plain roentgenogram of the abdomen often shows a markedly distended colon, with little or no evidence of small bowel dilatation. I have considered these findings an urgent indication for operation. 3. The third indication is the occurrence of continued or repeated bleeding, especially if hemorrhage is extensive enough to produce a shock state. This constitutes an urgent indication for surgical intervention. 4. The fourth indication is the presence of septicemia or metastatic infection, such as endocarditis, pyoderma, or arthritis, which must be treated by removal of the primary focus after administration of a preliminary course of antibiotics.

OPERATIVE TECHNIQUE

The technique of operation is simple and has become standardized. A left rectus incision facilitates mobilization of the splenic flexure and leaves the right side of the abdomen free for ileostomy. The colon is mobilized by freeing its lateral peritoneal attachments and detaching the greater omentum from the stomach. The mesenteric vessels are then ligated and divided, and the colon is removed to the level of the rectosigmoid. The terminal ileum is brought out through a stab wound in the right lower quadrant as a permanent ileac stoma. After the occurrence of two near fatalities from ascending ileitis, I adopted Dennis'[3] suggestion of doing frozen section biopsies of the terminal ileum and resecting enough so that the exteriorized bowel is microscopically free from disease. The divided rectosigmoid is exteriorized

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