POST-OPERATIVE CORONARY OCCLUSION

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In 1910 Osler[1] correlated the clinical and pathological findings in occlusion of the coronary arteries. Two years later Herrick[2] described the clinical syndrome clearly and really stimulated modern interest in the subject. Coronary thrombosis as a post-operative complication has apparently received very little consideration. Wilson[3] mentions one case of coronary embolism in a review of forty-seven cases of post-operative embolism. This followed a gastroenterostomy for chronic duodenal ulcer. Neuhof and Aufses[4] have recently studied the cause of death after operation in eight hundred consecutive autopsies in three New York Hospitals. In this series there is no reference to coronary occlusion. Balfour[5] reviewed fifty-one deaths following operations on the stomach and duodenum, one of which he has listed as due to coronary thrombosis. Herrick[6] mentions one death following cholecystectomy. Apparently the condition has not interested surgeons to any great extent. Even those surgeons who have discussed at some length "the acute abdomen,"[7,8] do not give acute coronary occlusion as a factor in differential diagnosis. Their medical confreres, however, have been keenly aware of its importance in the diagnosis of upper abdominal pathology, especially when it is to be differentiated from gall-stone colic, perforated gastric ulcer and acute pancreatitis. The "acute indigestion," so commonly spoken of in the lay press as a cause of death, is probably, in a high percentage of cases, due to coronary thrombosis.[9]

Following are reports of two cases of coronary occlusion following operation, occurring in the Surgical Clinic of the University of Kansas.

Case I.—H. A. V., male, age fifty-eight, was admitted to the Hospital March 25, 1916, complaining of a double inguinal hernia. There was nothing important noted in the past history, except attacks of palpitation of the heart. Positive findings on examination were overweight, artificial eye, pyorrhœa, chronic tonsillitis, irregular pulse with extra systole every three to six beats, blood pressure 160 systolic and 110 diastolic. The urine showed albumin and a few hyaline casts. There was a definite inguinal hernia on each side and hydrocele on the left. The blood count and hæmoglobin were normal.

With beta-eucain one-quarter of one per cent. as anæsthetic, a left herniorhaphy and hydrocele excision were done. Before the operation was finished, evidence of beta-eucain poisoning manifested itself by profuse perspiration, slow pulse, and slow irregular respiration. For twelve hours he had continued respiratory distress, slow, weak pulse and at times Cheyne-Stokes type of breathing. During this time he was given adrenalin and intravenous salt solution. He apparently recovered from the poisoning and the next day was making normal post-operative progress.

On the fourth post-operative day about 1:40 A.M., he raised himself slightly in bed to take a drink of water when he suddenly fell back on his pillow, took four or five stertorous gasps and died.

The pathological report by Dr. R. H. Major is in part as follows:

Heart: "On opening the pericardial cavity there is an excess of clear, straw-colored fluid present. The pericardium is smooth and glistening. The heart weighs 560 grams. There is a marked dilatation of the right ventricle. The wall of the right ventricle measures four millimetres in thickness. The wall of the left ventricle measures one and four-tenths centimetres in thickness. Heart valves are normal. The musculature on section has a distinct brownish appearance. The right ventricle shows numerous small, reddish, pin-point areas just beneath the pericardial covering. The aortic arch shows numerous small atheromatous patches. The opening of the right coronary artery just as it passes out of the aorta shows a complete plugging with yellowish white material. On dissecting out the right coronary artery the main trunk and small branches are found plugged with an embolus and the lumen entirely obliterated."

CASE II.—Y. P., a male, age sixty, was admitted to the Surgical Clinic of the University of Kansas December 19, 1929, complaining of pain in the epigastrium. Three months before admission he had received an injury to his upper abdomen by being suddenly jerked by the handle of a road grader. This caused him to have a sudden pain in his epigastrium, as if something had been torn. Since that time he has had more or less soreness and discomfort in the epigastric region, and has been unable to work. At the time of the accident he was somewhat short of breath but this has disappeared. He gave a history of an umbilical hernia for several years. There was some doubt about a small epigastric hernia following the injury. It was thought best, under the circumstances, to repair the umbilical hernia and explore the upper abdomen to determine if there was a small hernia through the fascia or a rupture of the rectus muscle. General examination revealed no other evidence of disease, except arteriosclerosis. His blood pressure was 95 systolic and 60 diastolic. There was a history of treatment for syphilis a few years ago.

December 20, 1929, a midline incision was made above the umbilicus and the upper abdomen explored. No definite hernia was found through the fascia, although there appeared to be some separation of the fibers of the anterior sheath of the rectus muscles. There was a definite diastasis of the recti muscles above the umbilicus. The umbilical hernia was repaired and the wound closed as usual. Following the operation, his condition was satisfactory except slight delirium which was attributed to amytal. He frequently attempted to get out of bed when there was no one to watch him. On the sixth post-operative day an infection was evident in his wound, with a temperature as high as 103 degrees. This was superficial and was easily drained, and the temperature dropped promptly.

On the eleventh post-operative day, he suddenly awoke with rapid respiration and gasping for breath. He did not complain of pain. The pulse was 100 and regular but rather weak. The blood pressure was 88 systolic and 50 diastolic. There was no cough or evidence of disturbance in the lungs. His color was ashen gray and he appeared quite ill. That day the temperature rose to 103 degrees. The respiration varied from 28 to 35 and was suggestive at times of Cheyne-Stokes type. His skin felt clammy. Examination of the heart and lungs did not reveal any definite pathology except distant, feeble heart sounds. The day following the onset of this trouble, a definite to and fro friction rub was discovered over the heart. The heart sounds were more faint than the day previous. His blood count showed a leucocytosis. A diagnosis of coronary occlusion was made. On the third day, the friction rub disappeared and was not heard again during his illness. For the first two or three days he appeared to be entirely conscious and answered questions rationally. Mental disturbance gradually developed. On the fourth day, following the accident, he became quite cyanotic with Cheyne-Stokes respiration and delirium. Abdominal distention was marked. He had a bilateral positive Babinski and ankle clonus at this time and it was thought he had developed emboli in his brain. On the sixth day it became evident that he was developing a hemiplegia. On one occasion he was quite maniacal and had to be held in bed. He had incontinence of fæces and urine. During his entire illness he had more or less cyanosis. A generalized

œdema developed which grew more marked till death. Evidence of general cerebral emboli gradually developed. His systolic blood pressure varied from 100 to 130. On various occasions electrocardiograph tracings showed nothing diagnostic. He gradually grew worse, and died on the fourteenth day following the accident. A final diagnosis of coronary occlusion and cerebral embolism with hemiplegia was made. Autopsy was not permitted. The immediate cause of death was apparently broncho-pneumonia.

The symptoms of coronary occlusion as given by Dr. Louis Hammon[10] are as follows: "1. The immediate symptoms associated with the occlusion; the anginal seizure: A. Pain; B. Shock; 1. Prostration; 2. Fall in blood pressure; 3. Suppression of urine.

"2. The symptoms associated with the myocardial damage, myocardial insufficiency: A. Dyspnœa; B. Passive congestion; 1. Cyanosis; 2. Pulmonary œdema; 3. Enlarged liver; 4. Albuminuria; 5. Subcutaneous œdema; C. Cheyne-Stokes breathing; D. Feeble cardiac impulse, faint heart sounds, gallop rhythm, murmurs, cardiac arrhythmias.

"3. The symptoms associated with the myocardial infarct: A. Fever and leucocytosis; B. Pericarditis; C. Embolic phenomena; D. Cardiac aneurysm and rupture.

"4. Additional symptoms: A. Nausea, vomiting, diarrhœa; B. Facies; C. Vasomotor symptoms; D. Nervous symptoms."

The close association between angina pectoris and coronary obstruction is well recognized and should aid in the diagnosis of the latter condition.

Discussion.—The two patients here reported were both males, fifty-eight and sixty years of age. Herrick[2] states that the condition usually occurs in men beyond fifty years of age with arteriosclerosis and previous attacks of angina pectoris. Both of our patients had definite evidence of arteriosclerosis, but neither gave any history of angina pectoris. In one case examination revealed an irregular pulse with what were apparently extra systoles and a blood pressure of 160 systolic and 110 diastolic. Blood pressure in the older man was 95 systolic and 60 diastolic and the pulse was regular.

In Case I the operation was done entirely with local anæsthetic. This was followed by definite evidence of beta-eucain poisoning and death occurred on the fourth post-operative day. In Case II one-half per cent. novocain was used followed by gas oxygen and ether. Wound infection followed. Preliminary to the anæsthetic he received 15 grains of amytal, ¼ grain of morphine and 1/200 grain of scopolamine. Evidence of coronary occlusion occurred on the eleventh post-operative day.

It has been stated by Evans[11] that post-operative thrombosis, in general, manifests itself most frequently at an interval of some days after the operation. Probably, if the tenth day be taken as a central point, a large proportion of the accidents occur within two or three days on either side of that point. He notes that the blood platelets rise following operation, reaching a maximum in about ten days and then declining to normal. After operation there is also a rise in blood-fibrinogen, apparent on the third day and persisting until the tenth day. The increase in these two important elements of clotting

following operation serves to explain why surgical patients may develop thrombosis or embolism about the tenth post-operative day. Our Case number II conformed to this rule. We see no reason why this explanation of Evans's should not apply to post-operative coronary thrombosis as well as such pathology elsewhere in the body.

We have no definite explanation to offer for the development of occlusion of the coronary arteries in these cases, other than possibly a thrombosis engrafted upon a preëxisting arteriosclerosis. The beta-eucain poisoning with its accompanying slow pulse and drop in blood pressure in the first case, and the infected wound in the second case, may have been contributing factors. These two cardiac accidents suggest the importance of a close pre-operative heart study, including a careful history concerning the occurrence of anginal symptoms. Such a history was not sought in our cases nor did the patient volunteer any such information.

It might be possible in certain cases to avoid post-operative catastrophe by taking a careful history of the heart condition and by making electrocardiographic studies, especially in patients over fifty years of age with arteriosclerosis. The work of Walters[12] should be noted when considering the prevention of thrombosis following operation. In order to combat the decrease of metabolism, the decrease in blood pressure and the slowing of circulation he has given tablets of desiccated thyroid gland in doses of two grains three times daily. With this treatment he reports a decrease in the frequency of pulmonary embolism.

Bancroft, Kugelmass and Stanley-Brown[13] believe that diet has a definite influence upon the clotting function of the blood. Mills[14] emphasizes the important relation of food intake to coagulability. Coagulability is increased with a protein diet and decreased with carbohydrates and fats. He calls attention to the fact that the platelet count rises just at the time most patients are allowed to begin mild exercise and take a full diet. There is a period when all factors favoring thrombosis are at a maximum; the platelets are greatly increased; increased protein intake increases their tendency to clump and disintegrate; this is further aided by the exertion and moving around, and finally the action of the sluggish circulation is intensified during the first few days of sitting up or getting out of bed.

CONCLUSIONS

In the pre-operative study and examination of males past the age of fifty, it is suggested that a careful cardiac history be taken to exclude angina pectoris as a predisposing cause of post-operative coronary occlusion.

A fall in the blood pressure following operation may be a contributing factor in the development of coronary thrombosis. Every effort should be made to prevent shock by proper selection of anæsthetic and operative technic.

Patients having any evidence of coronary disease should be treated with thyroid gland and careful regulation of diet until the danger period has passed.

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