canned goods, one sample of each brand available at retail at 4-monthly intervals for a year. If the adult consumes each day 2 servings of the indicated size of any one of the foods, or 1 serving of two of the foods, in addition to a mixed diet containing potatoes and a vegetable, he will be partaking of a daily diet supplying at least 65 mgm. of free ascorbic acid.

SUMMARY

A fasting blood plasma ascorbic acid level of 0.6 mgm. per 100 c.c. is considered by many investigators to be the lowest level consistent with normal ascorbic acid metabolism.

In recently reported studies subjects with a plasma ascorbic acid content of approximately 0.25 mgm. per 100 c.c. developed gingivitis more frequently than subjects whose ascorbic acid levels were above 0.75 mgm.

Study of the fasting blood plasma ascorbic acid levels of 372 young Canadians on admission to the Royal Canadian Air Force gave values below 0.6 mgm. per 100 c.c. in 55% and below 0.25 mgm. in 19%.

A marked change in the intake of ascorbic acid will reflect itself in the fasting blood plasma ascorbic acid content in a period of 1 to 2 weeks.

The effect of relatively constant levels of intake of free ascorbic acid over periods of 6 to 8 months on the fasting blood plasma ascorbic acid concentration was determined. With an average daily intake of 7.9 mgm. the average fasting plasma level was 0.22 mgm.; with an intake of 22.3 mgm., 0.26 mgm.; with 62.5 mgm., 0.75 mgm.; with 78.3 mgm., 0.87 mgm., and with an intake of 437.5 mgm., the blood plasma level was 1.49 mgm.

It is not only possible but relatively simple to consume meals that are attractive and palatable yet contain only in the neighbourhood of 5 mgm. of ascorbic acid per day.

While every effort should be made to conserve the ascorbic acid content of foods in their preparation, cooking and serving, a small number of foods if taken each day will supply the major portion of the recommended amount of ascorbic acid. These foods and the daily amounts required are set out in Table V.

REFERENCES

1. LINGHORNE, W. J., MCINTOSH, W. G., TICE, J. W., TISDALL, F. F., MCCREARY, J. F., DRAKE, T. G. H., GREAVES, A. V. AND JOHNSTONE, W. M.: *Canad. M. A. J.*, **54**: 106, 1946.
2. ZILVA, S. S.: *Brit. Med. Bull.*, **2**: 217, 1944.

RÉSUMÉ

On est d'avis que le taux de 0.6 mg. % d'acide ascorbique est le minimum normal que doit contenir le plasma sanguin chez l'individu à jeun. On a récemment rapporté que des individus ayant un taux d'acide ascorbique plasmatique de 0.25 mg. présentèrent plus fréquemment de la gingivite que ceux dont la concentration de cette vitamine dépassait 0.75 mg. 372 jeunes recrues de la R.C.A.F. avaient une concentration inférieure à 0.6 mg. % pours 55% d'entre eux, et de moins de 0.25 mg. % pour 19%.

L'ingestion de 62.5 mg. d'acide ascorbique par jour donnera un taux plasmatique d'acide ascorbique à jeun de 0.75 mg., ce qui correspond au taux convenable. L'ingestion d'une quantité moindre et d'une quantité supérieure donnera lieu à une diminution ou à une augmentation de ce taux selon le cas. Il est possible de prendre des repas très attrayants qui ne contiennent à peu près pas d'acide ascorbique. Une liste des aliments riches en cette vitamine figure au tableau V.

JEAN SAUCIER

DISSECTING ANEURYSM OF THE AORTA

(A Study of a Series of Fourteen Cases)

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A DISSECTING aneurysm of the aorta is a lesion produced by the extravasation of circulating blood through a rupture of the inner wall into the media. The extravasated blood extends for a varying distance, splitting the aortic wall into two layers. It is a relatively uncommon but distinct pathological condition which may give rise to a typical clinical picture. In many cases, however, the clinical features may simulate other acute conditions and because of the usual rapid termination, an ante-mortem diagnosis is often difficult. Death is usually caused by rupture of the outer wall of the aneurysm with a massive hæmorrhage. Occasionally a patient may survive and organization and obliteration of the aneurysmal sac or rupture of the inner wall of the sac back into the lumen may occur with the formation of a double-barrelled aorta.

The condition as known today was first described by Sennertus in 1628. The term dissecting aneurysm was first used by Laennec in 1826. A complete history is included in Shennan's[1] excellent monograph which appeared in 1934. He was able to collect 300 cases from the literature and to add 17 of his own which he reviewed in detail. Holland and Bayley[2] reviewed the American literature up to 1940. Occasional isolated

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TABLE I.

Case No.	Sex and age	Symptoms	Physical findings	Laboratory data
1.	M. 67	Sudden onset of severe pain in left upper quadrant of abdomen while in bed. Increasing dyspnœa and cyanosis. History of previous admission one month before with hypertensive cardiovascular disease and early congestive failure.	T. 98°, P. 80, R. 20, B.P. 230/160. Signs of moderate congestive failure. Heart 16.5 cm. to left of M.S.L. with systolic murmur at apex. Marked sclerosis of retinal vessels with small hæmorrhages.	Blood Wass. +++. Urine: + albumin and red blood cells present. Mosenthal showed impairment of renal function.
2.	F. 43	Sudden onset of severe pain interscapular region associated with headache, vomiting and blurring of vision. Angina pectoris five weeks.	T. 98.4°, P. 90, R. 22, B.P. 235/165. Heart 11 cm. to left of M.S.L. Early signs of congestive failure. Retinal hæmorrhages and papillœdema.	Blood Wass. Neg. Urine: albumin ++ White blood cell count 6,500. Blood N.P.N. and creatinine normal.
3.	F. 50	Sudden onset of sharp shooting pain in right upper quadrant of abdomen radiating back and up to the level of the right scapula. Vomiting and headache. History of rheumatic fever two years before death.	T. 99°, P. 70, R. 20, B.P. 162/80. Heart 10 cm. to left and 4 cm. to right of M.S.L. Auricular fibrillation. Apical systolic murmur. Obese. Icteric tinge to skin.	Blood Wass. Neg. White blood cell count 12,000. Hb. 67%. Van den Bergh 3.6 mgm. %.
4.	F. 29	No history of pain. Admitted to Maternity Pavilion 5 months pregnant with dead fetus. Nausea, vomiting and chills. Two previous admissions for hypertensive cardiovascular disease of three years' duration.	T. 99.4°, P. 80, B.P. 204/142. Heart enlarged. No murmurs. Uterus size of five months' pregnancy. Fundi normal.	Blood Wass. Neg. Urine: albumin ++ and hyaline casts.
5.	M. 29	Onset of aching pain left chest and precordial region while in hospital because of progressive congestive failure of one month's duration. Hæmoptysis two weeks. History of dyspnœa on exertion for one year.	T. 98°, P. 120, R. 20, B.P. 202/104. Heart 12 cm. to left. No murmurs. Signs of congestive failure present. Albuminuric retinitis.	Urine: albumin ++, hyaline and granular casts and red blood cells. White blood cell count, 6,100. Hb. 68%, N.P.N. 112 mgm. %.
6.	F. 69	Sudden sharp pain left chest radiating to both sides, worse on movement; radiated down back into thighs; responded partially to morphine; lasted two days. Recurrence similar attack six days later. Dyspnœa on exertion two years.	T. 98°, P. 100 irregular, R. 25, B.P. 130/80. Heart enlarged. Aortic systolic murmur. Upper abdominal tenderness. Slight amount of bloody fluid aspirated from chest.	White blood cell count 14,000. Hb. 70%. X-ray of chest: aorta widened.
7.	F. 50	Admitted in diabetic coma. History of diabetes nine years. Complained of pain in precordial region six days.	Obese. Kussmaul's respirations. B.P. 120/70. Heart slightly enlarged. No murmurs. Signs of consolidation both lung bases.	White blood cell count 12,300. Urine: ++++ sugar and acetone. CO_2 capacity 37%.
8.	F. 42	Sudden onset severe precordial pain associated with vomiting; lasting eight hours. Two similar attacks next four days. History of syphilis twenty years ago.	Blood pressure unobtainable. Heart enlarged. Arrhythmia. Signs of peripheral vascular failure.	Blood Wass. Neg. Chest x-ray showed enlargement of aorta.
9.	M. 63	Severe left upper quadrant pain radiating to back and downward to testicle. Intermittent for one week and associated with nausea and vomiting. History of hypertension.	Obese. Mild shock. B.P. 85/50. Heart enlarged. Systolic murmur at apex. Abdomen distended and some resistance left side.	White blood cell count 15,000. Urine: neg.
10.	M. 31	Sudden onset of right supra-orbital headache, left hemiparesis and weakness of right side of face. Shock. History of personality changes five months before admission.	T. 102°, P. 105, R. 20, B.P 100/72. Heart 10 cm. to left of M.S.L. No murmurs. Right hemiplegia.	Blood Wass. Neg. Urine: albumin ++ and hyaline casts. White blood cell count 12,000.

TABLE I.

Course and duration	*Gross pathology*	*Microscopic pathology*
Blood pressure persistently high. Died suddenly fifteen hours after onset of pain.	Aorta: intimal tear in transverse arch; dissection extended to the level of diaphragm. Outer coat of arch ruptured into left pleural cavity. Heart: markedly enlarged (800 gm.). Syphilitic ulceration on legs.	Aorta: medial necrosis and moderate intimal arteriosclerosis. Kidney: marked arteriolonephrosclerosis.
Blood pressure persistently high. Occasional mild precordial pain. Recurrence of severe pain 10 days after onset; sudden death.	Aorta: intimal tear 7 cm. distal to origin of left subclavian. Dissection extended from the base to the bifurcation of the aorta. Rutpure of outer coat opposite inner tear and into pleural cavity. Heart: enlarged (600 gm.). Ascites.	Aorta: marked medial necrosis. Heart: slight perivascular fibrosis. Kidneys: arteriolonephrosclerosis.
Pain persisted throughout stay in hospital. Sudden death six days after onset.	Aorta: intimal tear just distal to origin of left subclavian. Dissection involved the whole aorta down to and including the first portion of the left iliac. Outer wall ruptured into left pleural cavity. Heart: enlarged (500 gm.) Cholelithiasis.	Aorta: marked medial necrosis. Heart: slight perivascular fibrosis. Kidneys: marked arteriolonephrosclerosis.
Persistent nausea and vomiting. Sudden death seven days after admission.	Aorta: intimal tear at junction of transverse and descending portion of arch. Dissection involved arch and descending aorta to the level of the diaphragm. External rupture into left thoracic cavity. Arteriosclerosis present. Heart: hypertrophied and dilated. 100 c.c. of serous fluid in pericardium. Renal infarct. Five months' pregnancy.	Aorta: medial necrosis and arteriosclerosis. Heart: perivascular fibrosis. Kidneys: marked arteriolonephrosclerosis.
Development of progressive uræmia. Sudden death six days after onset of precordial ache.	Aorta: intimal tear in arch just beyond the origin of left subclavian. Dissection in descending portion of arch (4 x 2½ cm.). No external rupture. Marked arteriosclerosis. Heart: dilated and left sided hypertrophy (620 gm.). Lungs: bronchopneumonia and passive congestion. Kidneys: right 130, left 70 gm. Intracerebral hæmorrhage.	Aorta: medial necrosis and marked intimal arteriosclerosis. Kidneys: chronic pyelonephritis and moderate arteriolonephrosclerosis.
Persistent pain while in hospital. Right hemiplegia eighth day. Sudden death twelfth day.	Aorta: intimal tear in descending portion. Dissection involved arch and upper half of thoracic aorta. External tear into left pleural cavity and mediastinum opposite intimal tear. Moderate arteriosclerosis. Heart: enlarged. Moderately sclerotic mitral and aortic valves. Lungs: bronchopneumonia. Kidneys: hæmorrhagic infarction. Thrombosis of lt. int. carotid with cerebral infarction.	Aorta: medial necrosis and moderate intimal arteriosclerosis. Heart: perivascular fibrosis. Kidneys: moderate arteriolonephrosclerosis and hæmorrhagic infarction.
Responded poorly to treatment. Temperature persistently elevated. Died two days after admission.	Aorta: intimal tear in ascending arch. Dissection involved only ascending portion of arch (3 cm. in length). No external rupture. Moderate arteriosclerosis. Heart: 290 gm. Dilated and flabby. Lungs: lobar pneumonia.	Aorta: medial necrosis. Kidneys: a minimal arteriolonephrosclerosis.
Three attacks of pain while in hospital. Died during third attack four days after onset of illness.	Aorta: intimal tear in first portion of arch. Dissection extended from pericardium to diaphragm. External wall ruptured into pericardial cavity. Minimal arteriosclerosis. Heart: enlarged. Lungs: passive congestion. Cardiac tamponade.	Aorta: medial necrosis and intimal arteriosclerosis. Heart: patchy areas of degeneration. Kidneys: arteriolonephrosclerosis.
Died twelve hours after admission.	Aorta: irregular sacculated aneurysm in the abdominal aorta just below renal arteries. Intimal tear left side of aneurysm. Extravasation of blood into subperitoneal and left perirenal areas. Another aneurysmal dilatation filled with clot in left common iliac. Heart: enlarged, 515 gm. Marked arteriosclerosis of coronary vessels. Pulmonary congestion and œdema.	Aorta: medial necrosis. Heart: interstitial fibrosis. Kidneys: arteriolonephrosclerosis.
Day after admission onset of extreme dyspnœa, cyanosis and unconsciousness. Death second day of illness.	Aorta: intimal tear in ascending portion of arch. Dissection extended from the aortic valve to the right innominate artery into which it extended for 1 cm. External wall ruptured in two places, into pericardium and into mediastinum. Minimal arteriosclerosis. Heart: markedly enlarged. Lungs: passive congestion. Liver and kidneys: enlarged. Hypoplasia of bone marrow.	Aorta: medial necrosis, and minimal intimal arteriosclerosis. Heart: fibrosis and hypertrophy. Kidneys: minimal arteriolonephrosclerosis.

TABLE I.

Case No.	Sex and age	Symptoms	Physical findings	Laboratory date
11.	M. 55	Severe pain starting in the epigastric region radiating into chest and back.	T. 97.4°, P. 60, R. 25, B.P. 220/160. Heart enlarged. No murmurs. Local tenderness over sternum.	Blood Wass. Neg. White blood cell count 11,000.
12.	M. 64	Sudden onset severe pain between scapulæ radiating into abdomen and lower groin; started nine days before admission and constantly present. Associated with nausea and vomiting. Four years before death B.P. 154/96.	T. 98°, P. 120, R. 21, B.P. 235/175. Dropped to 180/70 just before death. Heart enlarged. No murmurs.	Blood Wass. Neg. Urine: albumin + + + +, hyaline and granular casts and red blood cells. White blood cell count 16,000.
13.	F. 44	Sudden onset of constriction, choking feeling in chest associated with weakness and dimming of vision. History of hypertension one year and for past three months dyspnœa and palpitations on exertion.	T. 96°, P. 55, R. 15, B.P. right arm 56/16, left arm 90/30. Heart enlarged. Systolic murmur at apex.	Blood Wass. Neg. Urine: trace of albumin. White blood cell count 12,000. Sedimentation rate: 48.
14.	M. 42	Sudden onset of severe sharp pain in precordial region lasting about three hours. Associated with dyspnœa and vomiting. Three similar attacks during twenty-four hours preceding admission. History of hypertension for three years.	P. 86, B.P. 90/60. Trachea deviated to the right. Widening of the mediastinum to percussion. Heart moderately enlarged. No murmurs.	Blood Wass. Neg. White blood cell count 9,600. X-ray of chest showed widening of superior mediastinal shadow.

cases have been reported and a few series have appeared since that time, such as those of Mote and Carr,[3] Shapiro,[4] Reich[5] and Glendy, Castleman and White.[9]

In spite of the number of cases which have been described, clinical recognition of this condition is infrequent. A review of autopsied cases during the past 20 years at the Royal Victoria Hospital showed that the diagnosis was seldom made clinically. It was hoped, therefore, that a detailed study of these cases might give some further information as to the etiology and also help to correlate further the clinical and pathological findings. In a twenty year period from 1926 to 1946, there were 14 cases of dissecting aneurysm of the aorta autopsied at the Pathological Institute. The total number of autopsies during this period was 8,048, which gives an incidence of 1 in every 575 autopsies. The incidence reported in the literature varies from 1 in every 175 as reported by Shennan[1] to 1 in every 552 as reported by Peery.[6] The average incidence is about 1 in every 380 necropsies.

The majority of the reports in the past showed that males were affected twice as frequently as females. In this series there were 7 females and 7 males. The average age was 49 years. The age distribution in decades was, two in the third, one in the fourth, four in the fifth, three in the sixth and four in the seventh. The males were slightly older, their average age being 51 years as compared to 47 years for the females.

The clinical picture was variable. Table I gives a summary of the main positive clinical and pathological findings. In all cases the onset was sudden and occurred while the patients were at rest. There was no history of any exertion precipitating the dissection. In three of the cases the dissection occurred while the patients were in bed in the hospital, being treated for another condition, one for toxæmia of pregnancy (No. 4), the second for diabetic coma (No. 7) and the third for congestive heart failure (No. 5). The most constant finding was the sudden onset of severe pain, usu-

TABLE I.

Course and duration	*Gross pathology*	*Microscopic pathology*
Sudden death one day after onset.	Aorta: intimal tear in distal part of transverse arch. Dissection along entire aorta involving the renal arteries and the right common iliac. The dissection ruptured into the left renal artery near its origin. External rupture in lower portion of thoracic aorta with hæmorrhage into left pleural cavity and into retroperitoneal tissue. Right renal artery occluded. Minimal arteriosclerosis. Heart: 450 gm. Right kidney infarcted.	Aorta: medial necrosis. Heart: hypertrophy and slight fibrosis. Kidneys: malignant nephrosclerosis. Infarction of right kidney.
Sudden death twenty-four hours after admission and ten days after onset of pain.	Aorta: intimal tear lateral to origin of left subclavian. Dissection extended to bifurcation of aorta. Left renal artery involved for distance of 1 cm. No external rupture. Minimal arteriosclerosis. Heart: hypertrophied, 530 gm. Lung: left lower lobe infarcted. Multiple cavernous hæmangiomas of the subcutaneous tissues.	Aorta: medial necrosis. Heart: slight fibrosis.
Low grade temperature first four days. Persistent dyspnœa and weakness but no pain. Progressive onset of congestive failure. Died 12th day.	Aorta: intimal tear in ascending portion of arch. Dissection involved entire aorta from the aortic valve to the bifurcation. Heart: hypertrophied and dilated (540 gm.). Orifices of coronary vessels and roots of innominate and left subclavian arteries involved by dissection. Lungs: hyperæmic and œdematous. Liver: passive hyperæmia. Ascites.	Aorta: medial necrosis. Aneurysm lined by endothelium and some organization of thrombus present. Cœliac, superior mesenteric, right renal and common iliac arteries showed medial necrosis. Heart: extensive fatty degeneration. Kidneys: arteriolonephrosclerosis.
Low grade temperature. Blood pressure improved to 130/80. Comfortable until 2nd day when pain recurred; hæmoptysis and death.	Aorta: intimal tear lateral to the left subclavian. Dissection extended downward to just below the level of the diaphragm. Outer wall ruptured opposite internal tear, into anterior mediastinum and both pleural cavities. Heart: enlarged (475 gm.). Right kidney: multilocular cyst upper pole (300 gm.).	Aorta: marked medial necrosis. Kidneys: chronic pyelonephritis and arteriolonephrosclerosis. Multiple encapsulated adenomata of both kidneys.

ally in the precordial region or in the upper quadrants of the abdomen, which radiated up into the precordium and later towards the back. In two cases (Nos. 6 and 9) the pain radiated downwards into the lower abdomen and in these the dissection involved the abdominal aorta. The pain was intense and only partially relieved by morphine. In some cases it was persistent and in others it recurred several times in severe attacks. Pain was absent in three cases. One of these patients (No. 14) experienced only severe dyspnœa. Another, (No. 9) suffered from a sudden onset of headache in the supraorbital region and extreme dyspnœa, following shortly afterwards by a left hemiplegia. Nausea and vomiting were the presenting features in another patient (No. 4). In eight cases the pain was associated with nausea and vomiting which were present before the administration of morphine. In three instances marked dyspnœa was the most common secondary symptom. All patients showed marked prostration, and in four cases (Nos. 10, 11, 14 and 15) definite symptoms and signs of shock were present.

The most constant finding on physical examination was the presence of hypertension. The blood pressure was elevated on admission and remained high in eight cases. In five cases the blood pressure was low on admission, but the past history and the pathological examination revealed definite evidence of hypertensive cardiovascular disease. In only one patient (No. 7) was there no clinical or pathological evidence of hypertension. Except for this one case, all patients showed definite enlargement of the heart. Three cases were in congestive heart failure. Systolic murmurs were present in several cases but there was no evidence of valvular disease. The pulse was regular and the rate only slightly elevated except for two cases, which had auricular fibrillations. There were no signs pathognomonic of dissecting aortic aneurysms. The positive findings were those of hypertensive cardiovascular disease and its results as shown in Table I.

The temperature was normal or only slightly elevated on admission. In all patients who survived for one or more days, the temperature after the first day rose to 100 or 101°, and remained elevated for three to four days, when it returned to normal. The white blood cell count and sedimentation rate were moderately elevated a few days after the onset of the dissection. The urine in most cases contained albumin, hyaline casts and occasionally red blood cells. The blood Wassermann was positive in only one patient (No. 1). The chest roentgenogram, showed widening of the arch of the aorta in three cases (Nos. 6, 8 and 14). The electrocardiogram, which was available in ten cases, showed only changes typical of marked hypertensive cardiovascular disease. A significant finding was that in the few cases, where the records were available, comparison of the electrocardiograms taken before and after the dissection revealed no changes. Repeat electrocardiograms in the cases which survived long enough showed no alterations except in one instance (No. 13), where changes suggestive of coronary occlusion appeared on the tenth day. The autopsy of this case showed involvement of the coronary orifices by the dissection.

The average survival was four and a half days after the onset of symptoms suggestive of dissection. The shortest was six hours, and the longest was twelve days. The course was variable. Ten patients died suddenly during a paroxysm of pain and collapse. A few of these had had persistent precordial pain, but the majority felt relatively well between recurring attacks of severe pain. One patient died of progressive congestive failure, one of uræmia and bronchopneumonia, another of pulmonary infarction and the fourth of diabetic coma, complicated by lobar pneumonia.

When the body cavities were opened usually a massive hæmorrhage and blood clot were found in the thoracic cavity (Nos. 1, 2, 3, 4, 6, 8, 10, 11 and 14). The left pleural cavity was the most common site of the intrathoracic hæmorrhage. The abdominal cavity contained blood in two cases (Nos. 9 and 11), and in the latter (No. 11) there was hæmorrhage in both the left pleural and abdominal cavities. In only two instances (Nos. 8 and 10) was there a hæmopericardium, which is much more frequent in other published series.[1] In four cases (Nos. 5, 7, 12 and 13) there was no external rupture of the aorta and death in these cases was due to cerebral hæmorrhage (No. 5), lobar pneumonia and diabetes mellitus (No. 7), massive pulmonary infarction due to an embolus in the left main pulmonary artery (No. 12), and myocardial failure due to external pressure on the walls of the coronary arteries by the dissecting aneurysm (No. 13).

The site and size of the intimal tear varied. In three cases, or 22.5%, (Nos. 7, 10 and 13) the intimal tear was in the ascending portion of the aortic arch, in six cases, or 43% (Nos. 1, 3, 5, 8, 12 and 14) it was located in the transverse portion of the arch, in four cases, or 28% (Nos. 2, 4, 6 and 11) it was found in the descending portion of the aortic arch or thoracic aorta, and in one case, or 7% (No. 9) the intimal tear was in the abdominal aorta. The location of the intimal tears differed from other reported series. Shennan[1] found the percentage distribution in the thoracic aorta to be 55% in the ascending portion of the arch, 16% in the transverse, and 8% in the descending and thoracic portion. Other authors give 70% as the frequency of occurrence in the ascending portion of the aorta.

The extent of the dissection was variable. The shortest was found in case No. 7, where it extended for 3 or 4 cm. The longest involved the entire aorta from its origin down to its bifurcation, and extended into one of the common iliac arteries (No. 3). The thoracic portion of the aorta was dissected to a varying extent in 7 cases (Nos. 1, 4, 5, 6, 7, 8 and 10). The thoracic and abdominal aorta were involved in three cases (Nos. 2, 3 and 11), and the abdominal aorta alone was dissected in only one case (No. 9). In three patients main branches of the aorta were dissected, the left common iliac (No. 3), the innominate artery (No. 10) and the left renal (No. 11). The site of the rupture of the external layer of the aneurysm was within 3 cm. of the intimal tear in eight cases (Nos. 1, 2, 4, 6, 9, 10, 11 and 14). In one instance (No. 3) the external rupture was farther from the intimal tear in the distal direction, and in another (No. 8) the external rupture was 6 cm. proximal, opening into the pericardium. In only one case (No. 11) was there a rupture back into the aorta, and in this instance it was through the left renal artery, but the aneurysm also ruptured externally. In

the entire series the aortas all showed varying degrees of intimal arteriosclerosis. In only one case (No. 9) was the intimal arteriosclerosis marked and extensive.

The microscopic examination of the aortas in all cases revealed degenerative changes, most marked in the media. The most prominent change in the media was a degeneration of the smooth muscle fibres, collagen bundles and elastic tissue fibres. The smooth muscle showed changes varying from cloudy swelling to complete degeneration with disintegration of the muscle fibres, and replacement by flocculent mucoid material. This same change was described by Shennan.[1] The collagen fibres also showed varying degrees of degeneration of the same type. In some aortas there were often found small discrete collections of fine basophilic granules in areas of degeneration. Where the elastic fibres were degenerated, the first change was a swelling and loosening of the fibres and then fragmentation. There were some large confluent cystic areas in many of the aortas, and here there was a degeneration and disappearance of all the elements of the media. These cystic areas appeared identical to those described by Erdheim,[7] and were located in the outer third of the media in the majority of cases. The inner third of the media usually showed the most extensive diffuse degeneration. A common finding in this inner portion of the media was a zone of collapse where the elastic fibres were in close approximation and the intervening muscle and collagen fibres had disappeared (Fig. 1). In one case (No. 14), a healed partial intimal rupture was found and described in the case report. The site of the dissection was always in the outer half of the media and this finding agrees with all other reports in the literature. Only in one case (No. 13) was there found any endothelial lining in the dissected cleft and this patient had survived for 12 days following her first symptoms.

The intima did not show any marked arteriosclerosis except in one case (No. 9). In this instance the intimal tear was through a large arteriosclerotic plaque in the abdominal aorta. However, medial necrosis was found in sections taken from the adjacent aorta. In all cases the adventitia was not remarkable except near the point of the external rupture, and here it contained extravasated blood. The vasa vasorum were not unusual and no pathological evidence of syphilitic aortitis was found in any of the cases.

Evidence of hypertension in the form of cardiac hypertrophy was present in every case except one (No. 7), in which the heart weighed only 290 grams. The kidneys in all cases, except one (No. 13), demonstrated varying degrees of arteriolonephrosclerosis. Other findings in the kidneys, heart and other organs may be seen by referring to Table I. In only one case (No. 13) was there extensive myocardial degeneration, and the reason for this, as has already been mentioned, was involvement of the coronary orifices by the dissecting aneurysm. The complete case report of a patient showing the typical clinical and pathological findings of a dissecting aneurysm of the aorta is given below.

CASE REPORT

A 48-year old white male was admitted to the Royal Victoria Hospital on March 11, 1946, by ambulance because of three attacks of severe precordial pain. At 6 a.m. on March 10, he was awakened by a severe sharp precordial pain associated with some shortness of breath and vomiting. The patient stated the pain was as if someone was driving a "screw driver" into his chest. It was partly relieved by morphine. The second attack of similar pain occurred at 4 a.m. and the third attack at 11 a.m. on March 11, the day of admission. Both attacks lasted about 3 hours.

The past history revealed that the patient had tuberculous adenitis (at the age of 18). Four years ago he was told he had hypertension and at that time suffered from symptoms suggestive of myocardial insufficiency. Ever since he had complained of dyspnœa on exertion. A month before admission he suffered from an upper respiratory infection which aggravated his dyspnœa and caused him to remain home from work.

Physical examination showed a thin, pale individual in no acute distress. The pulse rate was 86, but was weak and regular. The blood pressure was 90/60. The trachea was deviated slightly to the right and the movement of the chest was reduced. There were occasional râles at the posterior bases of the lungs. On percussion there was definite widening of the superior mediastinum. The heart was moderately enlarged, the sounds were distant but no murmurs were heard. The abdominal wall was slightly resistant, but there was no definite tenderness. The liver and spleen were not palpable.

The white blood cell count was 9,600 and the hæmoglobin 78%. The urine showed two plus albumin and many pus cells in the sediment. The blood Wassermann was negative. The electrocardiogram revealed a regular rhythm, a rate of 120 per minute and a low T wave in Lead I. A roentgenogram of the chest showed a widening of the mediastinal shadow with a marked bulge present on the right side. The aortic knobs could not be identified. The lung fields were clear.

Soon after admission he was put in an oxygen tent and given morphine with relief of the precordial pain. His temperature increased to 100° and his blood pressure increased to 130/80. He remained comfortable until 3.30 a.m. on March 13, when he again suffered from a sudden onset of sharp substernal pain and quickly passed into a state of shock and coma. He then expectorated mouthfulls of fresh blood and expired. The clinical diagnosis was dissecting aneurysm of the aorta.

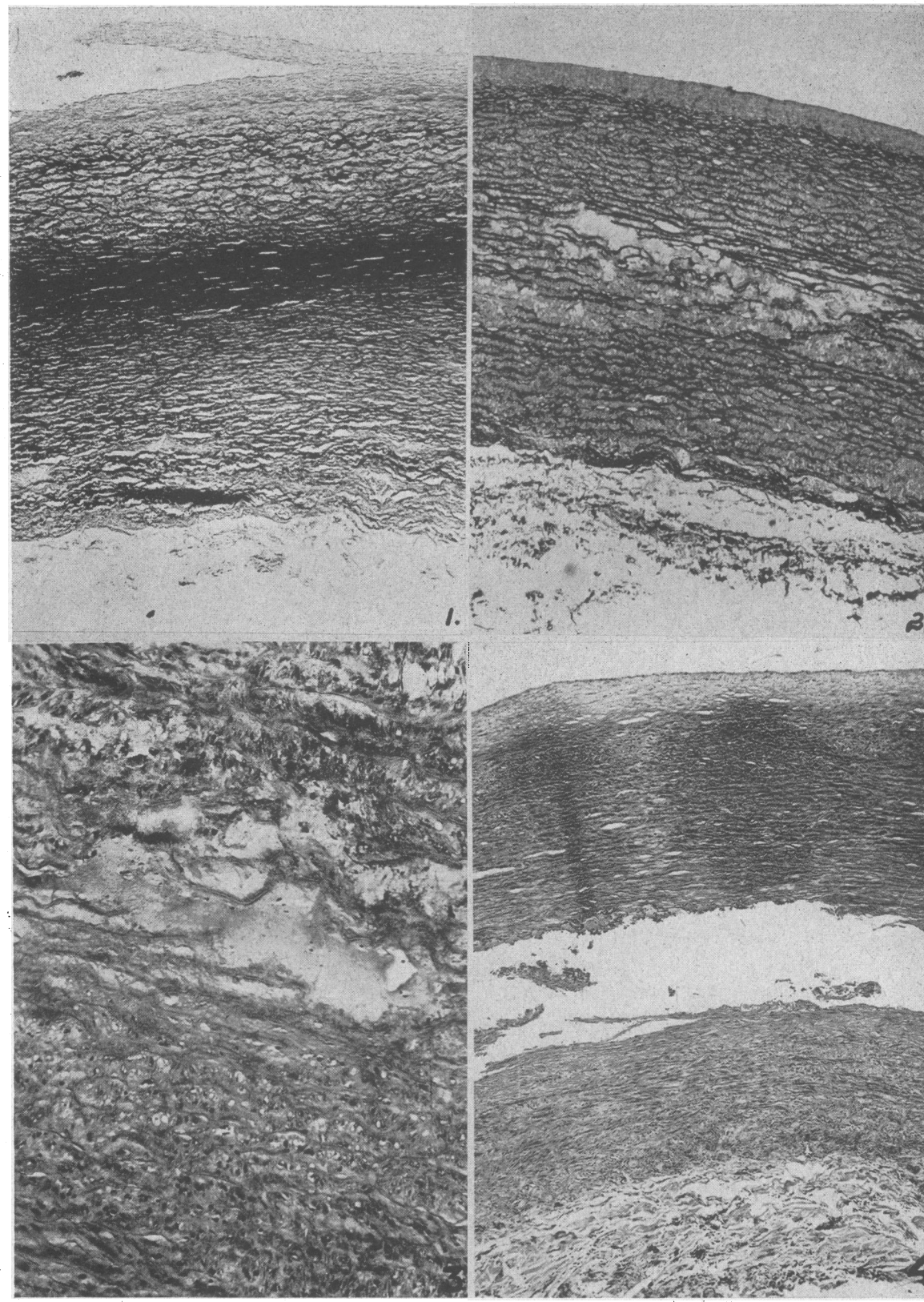

Fig. 1. (Case 14).—Section of wall of aortic arch with a well preserved intima and adventitia. The darkened area in the media is a zone of approximation of the elastic laminæ with a disappearance of the intervening muscle and collagen fibres. Verhoeff's elastic tissue stain. X 33. **Fig. 2.** (Case 14).—Section of the thoracic aorta showing cystic areas of degeneration in the media with fragmentation and disappearance of the elastic lamina, muscle and collagen fibres of the media in these regions. Verhoeff's elastic tissue stain. X 33. **Fig. 3.** (Case 14).—High power view of cystic area in the media of the aorta. Note the flocculent mucoid material surrounded by fragmented elastic fibres. Verhoeff's elastic tissue stain. X 117. **Fig. 4.** (Case 14).—Section of dissected area in the thoracic aorta. The split is in the outer half of the media. The intima and adventitia are well preserved. Hematoxylin and eosin. X 39.

Post mortem examination.—In the following summary of the autopsy findings only the relevant data are included.

The body was that of a well developed, emaciated white male. The thorax contained in all about 2,000 c.c. of fluid blood and clot. The anterior mediastinum and both pleural cavities contained blood, the right pleural cavity containing about 800 c.c. and the left pleural cavity about 500 c.c. of blood and blood clot. The upper lobes of both lungs were atelectatic in their posterior portions and emphysematous along their anterior margins.

The pericardial cavity was free from blood. The heart was enlarged and weighed 475 gm. There were no gross lesions in the endocardium, myocardium or coronary arteries. There was a minimal degree of sclerosis of the mitral valve. The circumference of the aorta was 8 cm. at its origin and a slight degree of elasticity was present in this region. There was a transverse tear in the intima measuring 1.5 cm. in length just lateral to the origin of the left subclavian artery. The tear opened into a dissecting aneurysm of the media which involved the posterior 2/3 of the circumference of the aorta and extended downwards to just below the diaphragm. The outer wall of this aneurysm was intact except for an area 2 x 3 cm. immediately opposite the intimal tear where rupture into the mediastinum had occurred. The intimal surface of the aorta showed a minimum degree of arteriosclerotic streaking. There were several depressed gray areas about 0.5 cm. in diameter in the intima of the ascending portion of the aortic arch.

The liver weighed 1,400 gm. and showed evidence of passive congestion. The spleen weighed 140 gm. The adrenals were of normal weight and appearance. The kidneys showed no gross evidence of nephrosclerosis.

Microscopic examination.—Sections from the arch and thoracic portions of the aorta showed a collapse of the central zone of the media with a disappearance of muscle fibres and their nuclei. Elastic tissue stains revealed the presence of closely spaced elastic fibrils. There were some areas in this necrotic zone of the media where small cystic spaces had formed which contained pale basophilic material (Figs. 2 and 3). These areas of cystic degeneration were more prominent in the arch of the aorta. A section from the edge of the dissection showed the media to be split and the aortic wall separated in the outer 1/3 of the media into two separate layers (Fig. 4). The abdominal aorta below the area of dissection presented marked and extensive disruption of the media, both the elastic and muscle fibres being fragmented. One of the areas of dimpling of the intima of the arch of the aorta proved to be a partial rupture with early healing underway (Fig. 5). Otherwise the intima and adventitia did not show any marked changes. The vasa vasorum appeared intact in all sections.

The liver showed evidence of a moderate degree of hyperæmia in the central areas of the lobules. The spleen also showed hyperæmia of its pulp and sinusoids. Evidence of chronic pyelonephritis and nephrosclerosis was present in both kidneys.

DISCUSSION

Although the clinical picture of these cases is variable, there are certain features which can be considered definite clinical manifestations of a typical case. The most characteristic feature was the sudden onset of severe precordial or upper abdominal pain, radiating towards the back and downwards into the abdomen in a person with known, or definite evidence of, hypertension. The pain differs in several respects from the pain of a typical case of coronary occlusion. It was always described as sharp or tearing, and never as constricting or crushing, and there was no radiation of the pain to the upper extremities. The pain also tends to be of maximum intensity right from

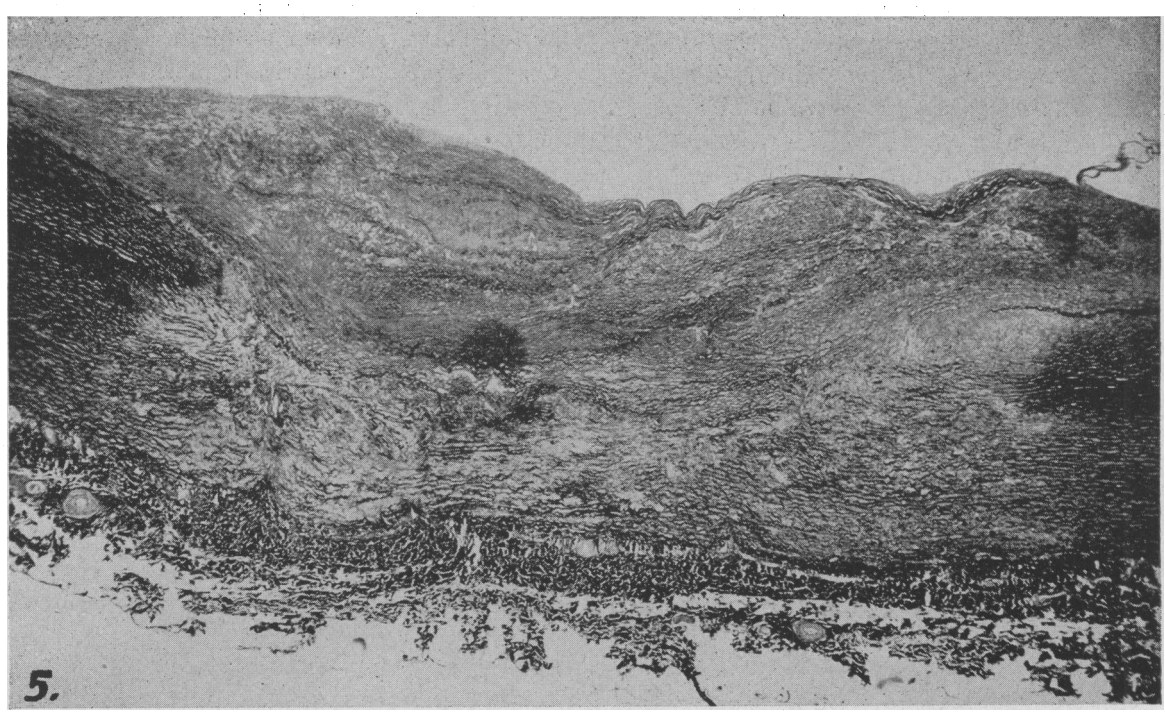

Fig. 5. (Case 14).—Section of a healed partial intimal rupture. Note the sudden disappearance of the elastic fibres in the media on either side of the tear with a thickening of the subintimal tissues by fibinous exudate, fibrous tissue, and fragmented components of the media. Hematoxylin and eosin. X 20.

the onset and more persistent and refractive to opiates. The presence of hypertension and the fact that the blood pressure remained high, even after the onset of the dissection, is an important diagnostic point. Another important feature is the absence of electrocardiographic changes suggestive of coronary occlusion in a patient with a history of the precordial pain described above. Roentgenogram of the chest showed suggestive widening of the arch in three cases. The importance of this sign in the diagnosis of dissecting aneurysm has been emphasized by Ritvo and Votta.[8] Symptoms and signs of blocking of the circulation to an important part of the body which White[9] describes as a helpful aid in diagnosis were found in only two cases (Nos. 6 and 10). Nausea and vomiting were common symptoms.

A definite ante-mortem diagnosis was made in one instance (No. 14) and strongly suspected in two others. The clinical diagnosis of coronary occlusion was made in all other cases. In the series of fourteen, only nine could be considered to present the typical clinical findings of dissecting aortic aneurysm. Three of these survived less than twenty-four hours after the onset of their illness. Unless a good description of the pain can be obtained from the patient, the diagnosis will be difficult and the more common condition of coronary occlusion will naturally be suspected. The negative electrocardiogram and suggestive roentgenological findings are only of value in a patient who survives for a period of several days. The increase in temperature, white blood cell count and sedimentation rate are common to both conditions. In three instances, already mentioned, where the dissection complicated another condition, or in one case (No. 9) where the symptoms suggested a surgical emergency, recognition would be impossible. A study of these cases therefore, reveals that it is a difficult diagnosis to make clinically. Heightened awareness might however increase the frequency of its proper recognition.

An analysis of the pathological findings in the series presented, demonstrates that the etiology of dissecting aortic aneurysm appears to be based largely on three factors: (*a*) medial necrosis, (*b*) hypertension, (*c*) arteriosclerosis.

The medial necrosis is probably the most important factor because without it we found no cases of dissecting aortic aneurysm. Many histologists consider that of the elements of the media the elastic lamina are the most important constituents, and that the other elements are attached to them. The smooth muscle fibres pass obliquely between elastic lamina and arise and insert into their covering connective tissue. The outer third of the media contains the largest amount of the smooth muscle cells.[11] When the aorta dilates during systole the tone of the muscle allows a gradual expansion of the aortic wall, and when the muscles are fully stretched they contract aided by the tension of the elastic laminæ. When medial necrosis occurs it affects first the smooth muscle and collagen elements and when these have degenerated the aortic wall will distend during systole, but will not contract. Thus the aorta becomes permanently dilated. The intima now becomes stretched, thinned and weakened, and sometimes there is a partial tear in the intima with healing. Usually the intima gives away completely to allow the blood from the lumen to penetrate the media where it seeks the line of least resistance, which is in the outer third of the media. The outer third of the media often contains cystic areas of degeneration where all the elements are degenerated. The prevalence of the extensive areas of degeneration here is probably because of the greater proportion of smooth muscle fibres. A common finding in our series was a zone of elastic laminæ which were closely approximated, with the disappearance of the intervening collagen fibres and muscle cells. This zone was always in the inner third of the media and can be attributed to the dilatation of the aortic wall, causing stretching and permanent approximation of these elastic laminæ. This is due to the degeneration and disappearance of smooth muscle cells which normally aid the elastic fibres to contract. The site of the intimal tear was in some part of the aortic arch in nearly all our cases. This is likely due to the fact that the highest intra-aortic pressure, and hence greatest strain, is put on this portion, both during systole and diastole.

Medial necrosis of the aorta has been produced experimentally in rabbits by several investigators using the intravenous injection of diphtheria toxin. Klotz[12] in 1906 was one of the first to produce these lesions and later Bailey[13] and then Duff[14] were able to produce

similar lesions. Intravenous tyramine injections given to rabbits, as used by Duff, Hamilton and Magner[15] produced medial necrosis of the aorta in some of their animals. Most investigators think medial necrosis is a degenerative condition due to the accumulated effects of various toxins and perhaps accentuated by the natural aging processes or dietary deficiencies, or both, as suggested by Klotz and Simpson.[16] In one patient, not included in our series, marked medial necrosis with no dissecting aortic aneurysm was found. He was a 50-year old male who was admitted to the Royal Victoria Hospital with furunculosis, staphylococcus septicæmia and bacteræmia and he died after 4 days in hospital. At autopsy, in addition to other findings, a fusiform dilatation of the ascending portion of the aortic arch was found. The microscopic examination of the aorta revealed marked medial necrosis. This, then, was a case where a severe toxæmia and medial necrosis co-existed and it is interesting to speculate whether the toxæmia was an etiological factor in the medial necrosis. The absence of dissecting aneurysm may have been due to the lack of hypertension. From our series it would seem quite definite that neither syphilitic infection nor other acute or chronic inflammations played any part in the causation of the necrosis of the media.

The second factor in the etiology of dissecting aortic aneurysms in our series is hypertension, and this condition was present in all cases but one. The rupture of the intima, dissection of the media and rupture of the adventitia are all probably initiated by the increased intra-aortic pressure as is present in hypertension. The kidneys in every case but one showed varying degrees of arteriolonephrosclerosis. Other authors have emphasized the importance of chronic kidney disease as a link in the chain of events.

The third factor of arteriosclerosis in the intima is not so important in our series. It did exist in all cases to a varying degree, but this would be expected in this age group of patients. In only one instance could arteriosclerosis be incriminated as a major etiological factor.

Although other authors[1, 16] consider that the presence of coarctation of the aorta is important in determining the site of the intimal tear, no anatomical abnormalities on the basis of congenital defects were found in any of our cases.

SUMMARY

1. The clinical and pathological findings in 14 cases of dissecting aneurysm of the aorta have been presented.

2. The sudden onset of severe sharp pain in the precordial or upper abdominal region radiating to the back and sometimes downwards into the lower abdomen in a patient with evidence of hypertension is a characteristic clinical finding.

3. The pathological examination revealed two important etiological factors, medial necrosis and hypertension, and one factor of less importance, namely, intimal arteriosclerosis.

4. The reasons for its infrequent diagnosis have been discussed. It is felt heightened clinical awareness would increase the incidence of correct recognition.

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REFERENCES

1. SHENNAN, T.: Dissecting Aneurysms, H.M. Stationary Office, London, 1934.
2. HOLLAND, L. F. AND BAYLEY, R. H.: *Am. Heart J.*, **20**: 223, 1940.
3. MOTE, C. D. AND CARR, J. L.: *Am. Heart J.*, **24**: 69, 1942.
4. SHAPIRO, L.: *Canad. M. A. J.*, **49**: 408, 1943.
5. REICH, N. E.: *Clinics*, **3**: 346, 1944.
6. PEERY, T. M.: *Arch. Int. Med.*, **70**: 689, 1942.
7. ERDHEIM, VON J.: *Archiv. Fur Klinische Medizin*, **273**: 454, 1929.
8. RITVO, M. AND VOTTA, P. J.: *Am. J. Roent.*, **52**: 583 1944.
9. GLENDY, R. E., CASTLEMAN, B. AND WHITE, P. D.: *Ann. Int. Med.*, **11**: 845, 1937.
10. WILLIUS, F. A. AND GRAGG, R. W.: *Proc. Staff Meet. Mayo Clin.*, **16**: 63, 1941.
11. COWDRY, E. V.: Textbook of Histology, Lea & Febriger, Philadelphia, 1934.
12. KLOTZ, O.: *J. Exper. M.*, **25**: 109, 1917.
13. BAILEY, C. H.: *J. Exper. M.*, **25**: 109, 1917.
14. DUFF, G. L.: *Arch. Path.*, **13**: 543, 1932.
15. DUFF, G. L., HAMILTON, J. D. AND MAGNER, D.: *Proc. Soc. Exp. Biol. & Med.*, **41**: 295, 1939.
16. KLOTZ, O. AND SIMPSON, W.: *Am. J. M. Sc.*, **184**: No. 4, 455, 1932.

Public health experts realize that our science may be used either to save life or to destroy civilization. Whether science is to be used for good or for evil is not determined by scientists themselves. The same type of research worker may discover penicillin or atomic fission. It is the mass conscience of mankind—the dominance of the moral or the amoral—which determines whether research is to be used for life or death.—Thomas Parran.