Let us prepare our minds to join quickly in furtherance of a sound plan when it is presented.

Let us stand ready to throw our weight and resources of knowledge behind that plan.

Let us be ready to mobilize with alacrity.

Above all, let us select leaders whom we can trust—and then trust them.

350 Post Street.

SUPERIOR MESENTERIC THROMBOSIS

REPORT OF CASES

By WILBURN SMITH, M. D.
Los Angeles

DISCUSSION *by John Homer Woolsey, M. D., San Francisco; Thomas O. Burger, M. D., San Diego.*

SUPERIOR mesenteric thrombosis was practically unknown until that brilliant German pathologist, Virchow, discovered and described a postmortem case of a superior mesenteric artery enlarged, and so completely closed with a thrombus that it appeared as a fibrous cord. The patient, a woman, had succumbed to some other disease, and he wrote in detail of the case in 1847. The clinical side is not reported, for the condition had existed for some time, and nature had established a collateral circulation. This type of case usually passes unrecognized and is often entirely overlooked, being diagnosed as one of colic, or some partial intestinal obstruction. The patient may recover and succumb to some other disease. A postmortem may never be made, and should one be made the examiner may not be thorough enough to examine all the tissues, as did the brilliant Virchow, and so the condition never be discovered.

This condition is not common, for since the first careful description in 1847 about five hundred cases have been described, with only thirty-five of these surviving the attack whether operated or not. This gives the appalling mortality of 93 per cent. The artery is involved about five times as often as the vein, and the superior mesenteric is involved about forty times more often than the inferior. It is true the amount of intestine supplied by the superior is much greater, as it extends from the duodenum to the anastomosis with the inferior at the middle colic. Also Litten maintains that the superior is a type of end-artery and has more of a tendency to favor an infarct, while the inferior tends to establish a collateral circulation. One would suppose in an artery which forms arcades that collateral circulation would easily and most frequently obtain. Even though Virchow,[1] Karcher,[2] Chiene,[3] and others have discovered cases in the postmortem room that had died of other more marked pathology, yet collateral formation is the exception in this artery. Karcher's case was in a woman, forty-one years of age, who had cardiac decompensation symptoms with abdominal pain. She developed femoral thrombosis, and in six weeks was operated upon for gangrene of the leg. She died a week later and on postmortem showed, in addition to disease of the mitral, tricuspid and aortic valves, lateral thrombi in both auricles, infarcts in lungs, spleen and kidney, an obliterating thrombus in the profunda femoris and, what is more interesting, a thrombus obliterating completely the superior mesenteric artery for a distance of thirty-seven millimeters. This thrombus was firmly adherent to the walls of the artery, but in spite of the same there was only slight reddening of the mucous membrane of the ileum. Chiene's case showed an aneurysm in a woman sixty-five years of age with the celiac axis, superior and inferior, involved; and the latter vessels were completely obliterated, forming fibrous cords.

The causes of this rather rare condition may be better studied if we separate the pathology of the artery from that of the vein. Under the artery we think first of embolus which is often followed by thrombus, or of thrombus alone. The embolus comes from heart valves and vegetations, from atheromatous plaques, and from the breaking-up of a thrombus in the auricles or ventricles. The thrombus arises from diseased arteries, from aneurysm with extension of the clot, and from pressure on an artery due to an aneurysm or a tumor. In venous involvement various causes which may injure the veins, or infect them, or a combination of the two, are the factors. The more frequent causes are crushing and ligating of the appendicular veins, pelvic surgery where adhesions are present, splenectomy, volvulus, intussusception, strangulated hernia or extension from the splenic or portal veins. Clinically arterial and venous thrombi differ in that the arterial thrombus disposes to be sudden in onset, while the venous tends to be gradual.

EXPERIMENTAL WORK

Much experimental work has been done to ascertain the exact pathology and account for the variety of clinical symptoms manifest in these cases. Sprengel's theory that obliteration of an artery gave an anemic infarct, while the same in a vein gave an hemorrhagic one, does not here obtain; for, regardless of the cause, the infarct disposes to be hemorrhagic. This hemorrhagic infarct is usually followed by peritonitis, the mucous membrane ulcerates and breaks down with hemorrhage into the canal. The mesentery becomes edematous and the intestine may perforate and cause the peritonitis from macroscopic lesions. Extensive gangrene may develop in forty- eight hours and there may or may not be a distinct line of demarcation.

Following Litten's suggestion that the arteries are terminal, various experiments, namely, ligation of the artery or vein; making of artificial emboli by oil which is not typical and so not parallel; cutting portions of the mesentery along the intestinal attachment to study the effect on

the mucous membrane, have been done upon the lower animals, but whether the conclusions are safe to accept for the human is a question.

The result of the experiment of ligating the artery was anemia followed by violent tetanic contractions of the small intestine, followed in two or three hours by relaxation and a congestion which terminated in a hemorrhagic infarct. Beckman and Ravenna showed, in operations on rabbits, dogs, and cats, that no effect was produced and that collateral circulation was established. On injection of paraffin, a large infarct was produced the center of which tended to be anemic and the periphery hemorrhagic. Tying the mesentery along the border of the intestine in three-centimeter lengths produced first the change in the mucous membrane of areas of necrosis, while five-centimeter lengths produced necrosis in greater extent, first of the mucous membrane and later of the wall itself.

The deductions from the various experiments are as follows: Lodgment of embolus may not produce infarct and a collateral circulation may be established; slow closure by a thrombus may stimulate collateral circulation; many cases of closure of the artery may be overlooked, due to the collateral anastomosis, and the closed artery may escape detection at autopsy, since the cause of death is foreign to this pathology.

Welch and Mall ligated the collateral circulation of the small intestine at the pancreaticoduodenal and at the middle colic and nothing happened, as the superior mesenteric was still intact. They then compressed this artery and when it reached one-fifth of normal they began to get an infarction. This led them to believe it was a matter of pressure in the artery, whether due to pressure constricting the vessel or cardiac; namely, vis a fronte.

CASES FROM LITERATURE WHICH CORROBORATE THESE DEDUCTIONS

Karcher's case seems to corroborate the latter. A woman, forty-one years of age, with cardiac failure and collapse, was brought into the hospital. There was violent abdominal pain and frequent bloody stools, with distention of the abdomen and tenderness. The left leg was very painful along the femoral and later had to be amputated, due to gangrene. Heart decompensation continued and death followed a week after the amputation. The autopsy showed multiple lesions in the heart with infarcts in lungs, spleen and kidneys, and thrombosis of the femoral vein with obliteration of the superior mesenteric. Abdominal pain was due to lodgment of the embolus with injury to the intestinal mucosa, as evidenced by the bloody stools, but collateral circulation was fairly established. Many cases have cardiac lesions that have so lessened vis a fronte and decreased circulation, that venous stasis limits the blood passing through the part and lessens the ability to form collateral channels.

Councilman's[4] case was incomplete obliteration with fecal vomiting and obstipation and death from intestinal obstruction. Here the pathologist reported atheroma, blocking incompletely the superior mesenteric artery with no changes in the intestines. So the circulation may be partially disarranged with intestinal obstruction and the intestine still remain normal. Thus it requires more blood to keep the peristaltic function intact than it does to keep the life of the tissues. This conclusion is supported by intermittent claudication where the function is disturbed before the life of the tissues.

Reich's[5] case gave all the symptoms of intestinal obstruction; was operated for the same and, after careful exploration, nothing was found. The symptoms persisted and a colostomy and ileostomy was later done for the same, but the patient died. The autopsy findings showed arteriosclerosis of the aorta, partial thrombosis of the superior mesenteric, and an infarct involving the jejunum to the extent of eighty centimeters. No. doubt the thrombus existed and produced the ileus of this loop with the clinical symptoms, though the condition was not apparent at the time of operation.

In the above cases we see three very distinct classes of cases: (1) Karcher's, with other pathology overshadowing the thrombus and with the collateral circulation becoming established. (2) Reich's, the opposite extreme, with marked infarction and destruction of the mucosa. (3) Councilman's, intermediate, with intestinal obstruction the overshadowing symptom, and no marked pathology in tissues. The amount of pathology will depend on the cardiac compensation and the vis a fronte.

VALUE OF HISTORY

A careful history is of value in about two-thirds of the cases, and will show some suggestion of an etiology, namely, valvular disease, arteriosclerosis or aneurysm, with exciting causes of abdominal surgery on stomach, appendix, or hernia. There may be exertion, but pregnancy is also a factor. In the balance of the cases the history has no bearing. Cases in men are twice as frequent as in women, and occur between twenty and sixty years of age.

SYMPTOMS

Pain is constant in type and paroxysmal, wave-like, merging into the continuous pain of peritonitis. The intermittent type of pain may be ascribed to the anemia which for the first few hours is tetanic in type. As it becomes intermittent it simulates the pain of intermittent claudication. There may be some association with peristalsis disturbed by the obstruction.

Vomiting is frequently present, at first reflex, then obstructive, and later due to the ileus of peritonitis. This is of the stomach contents and in the severe pathology, of the intestinal contents, which will be eventually bloody.

Constipation or obstipation is present in two types of cases, namely, those with the severe destruction of the mucosa and those where peristalsis is paralyzed without destruction of mucosa. There may be slight amount of flatus with enema, but if the involvement is marked none will pass. Blood is present in the more severe form and will be present in about 41 per cent. Early diarrhea may precede the constipation. The constipation may at first be due to the paresis, or in turn to the gangrene, and later be followed by that due to the peritonitis.

The temperature may at first be subnormal but rise later, due to the peritonitis. The abdomen is tender throughout, but may be accentuated in a certain area. Gradual distention appears which is usually tympanitic, but may be flat in the sides later, due to fluid.

Palpation may reveal a local mass due to the edema in the mesentery, and if palpated, with the other symptoms and findings, helps one to make a diagnosis. On auscultation one finds a gradually decreasing amount due to the paresis, and the onset of the peritonitis.

A leukocytosis is usually present, approximately 20,000, with a differential of above 85 per cent polymorphonuclear leukocytes.

The symptoms referable to the pathology: (1) In very severe destruction the main symptoms are diarrhea with bloody stools and hematemesis, associated with collapse. (2) In less severe, all the symptoms and landmarks are of obstruction. (3) Combinations of the above, namely, bloody stools and hematemesis, with obstruction symptoms, may occur. (4) In cases with a meager pathology but with symptoms overshadowed by greater pathology the patient may form collateral circulation. (5) With very mild pathology, the symptoms are akin to ulcer, namely, pain following eating, which is relieved by vomiting. The pain is explained by the food stimulation of peristalsis, and this pain is like the intermittent claudication due to the anemia, and a partial ileus exists. This is followed by vomiting and relief. With this may be associated cardiac disease and hence low pressure in the superior mesenteric artery. This is also seen in splanchnic sclerosis where there is a decrease in the blood supply.

Gerhardt [6] says a typical case should present the following: A definite cause for an embolus, intestinal hemorrhage, paroxysmal pains, ileus, fluid in the abdomen, subnormal temperature with a palpable abdominal mass. If there is no vomiting of blood or diarrhea with bloody stools, then superior mesenteric thrombosis will resemble Councilman's case and be diagnosed intestinal obstruction.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis from the following are to be thought of: acute perforative appendicitis, volvulus, intussusception, and intestinal obstruction. The more severe cases of thrombosis with the manifestation of bloody stools and vomiting are not confused with the appendix. In the latter the pain is about the umbilicus, with initial vomiting and then temperature; in the former the pain passes to the right iliac fossa; there is no obstipation and a return of the vomiting is much later.

Volvulus occurs in older people with no specific etiology of thrombus, no bloody stools, but obstipation and distention and vomiting tend to place it as an intestinal obstruction. This in turn may be the points in the middle class of the thrombus where all symptoms, as in Councilman's, are obstruction.

Intussusception is more nearly like the severe type of thrombus. Here we see more often a child with sudden attack of obstruction with vomiting (not bloody), with bloody stools and often the sausage-shaped tumor, palpable per abdomen or (the head) per rectum. If there be no diarrhea of blood, and hematemesis, the cases are diagnosed as intestinal obstruction.

PROGNOSIS

The prognosis depends on the extent of the thrombosis and the early recognition and treatment. The progress is rapidly downward, with rising temperature, collapse and peritonitis. Sixty per cent die in the first week and the mortality approximates 95 per cent. Five hundred cases are reported in the literature and only thirty-five survived.

TREATMENT

Treatment may be classified according to the degree of the pathology.

In the very mild, the symptoms are ulcer with the pains as recited above, which are due to the peristalsis being excited by the food intake, and due to the anemia of the part, there is spasm. Here diets easily digested, with medication to assist digestion. The pathology is a partial closure or a very decreased blood supply due to splanchnic sclerosis.

In the more severe, with overwhelming other pathology, the abdominal symptoms are not of sufficient severity to warrant a laparotomy with the other cardiac, or cardiovascular symptoms, and here eventually, under symptomatic treatment, the collateral circulation is reëstablished.

In the very severe types where infarct forms, operation is the only method of choice.

REPORT OF CASES

Case 1.—At this point I wish briefly to describe two cases. The first was during my intern days, and was so unique that it was not easy to forget. A woman, approximately twenty-eight years of age, came into the hospital with only the symptoms of a chronic appendix. She was of the upper strata of society and could have everything she desired. My chief, one of the best surgeons I have ever known, operated and removed a small chronic appendix and there was no other pathology. The following days were tragic. Consultations were frequent. The patient began to vomit slightly, the abdomen to distend, and the bowels became more and more constipated. The tem-

perature rose gradually, and the abdomen became more silent until all the symptoms were of a peritonitis. The father was a prominent citizen and every means to quiet the vomiting was exhausted. Consultants had only suggestions of no avail; the eventual came. The postmortem revealed a thrombus in the ileocolic vein and a portion of the ileum was gangrenous with a diffuse peritonitis. I can see the patient, a reasonably healthy young woman, restless, vomiting, distended abdomen, nothing relieving the same and the anxiety of the relatives. It certainly impressed me that a chronic appendix is not to be despised nor a suppurating appendix to be despaired.

* * *

Case 2.—The next case, a nurse, thirty-two years of age, single, was admitted to the hospital complaining of pain in the right side. She had a negative urine, blood, and Wassermann. At this time, September 21, 1927, I removed her appendix and did a subtotal hysterectomy for a multinodular fibroid uterus. She was brought into the hospital almost four months later with a very acute abdomen, which had its onset about fifteen minutes after she had eaten a ham and lettuce sandwich. Her temperature was 97 degrees upon admittance, pulse of 64, and respiration of 20. On the 19th of January, the day following the admission, I saw her for the first time. She was now transferred to the surgical service. She gave a history of nausea and vomiting, severe paroxysmal attacks of pain, though it was more or less continuous.

The pains on admission were much more severe and the patient rolled in agony at the time. The admitting physician could not account for the severity of the pain, and being in a woman he feared it might be exaggerated. Since I had removed the uterus and appendix only four months prior, and there was no history of ulcer, I was firmly convinced there was a relation to the food, even though friends had partaken of the same kind of sandwich at the same time. Enemas were followed by a great discharge of blood, and the results of the enemas were not satisfactory as far as flatus and feces. The tenesmus and also the vomiting continued. The patient assumed a dorsal or lateral position, with the knees flexed, and the hands on the abdomen, in great pain. She had received an opiate under the medical care, so was not in the severe pain of the day previous. Her face was anxious and she looked haggard and tired. The heart was negative for valvular pathology; blood pressure of 138-70. The abdomen was distended and tympanitic, with a suggestion of flatness in each flank. Definite rigidity existed of the lower abdomen, possibly more pronounced on the left rectus. My impression was gastro-enteritis from the sandwich. A urine and blood examination was suggested, and this was later phoned to me as follows: 20,400 leukocytes, with 85 per cent polymorphonuclears. Urine 1.026, with one per cent indican. The urine showed the dehydration with a suggestion in the indican of small intestine trouble. The temperature was 99.2, pulse 76, respiration 20 in the morning, but in the evening it was 99.6, 84, and 22.

On January 20 the abdomen showed more tympany with less peristalsis, and only in the upper abdomen was there audible peristalsis. The lower abdomen was more tender and more rigid. A pelvic examination showed the cervix mobile and no evident tenderness in the cul-de-sac or the adnexal regions. The evidence was not conclusive, as the abdomen was too tender and rigid to allow abdominal palpation. A repetition of the enema gave blood and practically no flatus. A repetition of the blood count gave 20,450, with 86 per cent polymorphonuclears. A special urine was examined for indican and showed 1.030 specific gravity with a three plus indican. Temperature, pulse, and respiration in the morning were 99-100-16, and later in the day the temperature, pulse, and respiration were 99.6-100-18.

Because of the severe pain, followed by vomiting, which was marked at first but less the second day with continuing nausea; the unsatisfactory results with enemas and the presence of blood; the white blood count persisting and, if anything, a little increasing; and the marked increase in the indican, I made a tentative diagnosis of incomplete obstruction of the small intestine probably associated with the appendix operation. My reasons for the diagnosis were that appendicitis, salpingitis, and ectopic pregnancy were eliminated by the first operation. The paroxysmal severe pain associated with peristalsis, and the apresence of indican in excess, suggested small intestinal obstruction while the passage of the blood and slight flatus made me modify to incomplete obstruction.

Upon the above diagnosis I made a right rectus incision and removed the old scar, believing the trouble would be due to adhesions from the previous operation. On reaching the peritoneum it was dark, like that seen in a recent ectopic rupture, and on entering the abdomen free blood was present. It could not be ectopic, and I had never seen an ulcer give free blood and there were no food particles. I made a culture of the blood, which had a slight odor. On examining into the left pelvis I found a few recent adhesions trying to wall off a loop of ileum about fifteen inches long, absolutely black, and the endothelium losing its sheen and gloss. There was no evidence of bands nor volvulus, but the mesentery was thick and edematous and this blackness faded into the edema of the normal intestine. The vessels were thrombosed and the extent of the pathology was uncertain. The loop was withdrawn and excised and the ends of the ileum sutured into the lower angle of the wound. Plenty of drainage was inserted, a wide sheet of rubber tissue placed to wall off the upper from the lower abdomen, and the wound closed. A tube was inserted into the proximal and the distal loops of the bowel.

The patient made an uneventful recovery with an ileostomy wound. We used the distal end to give Murphy drip and thus save the patient from nausea incident to the need for excess fluids. In about four weeks I again operated and did an end-to-end anastomosis. The patient had a good recovery and left the hospital March 14 with only slight soiling of the dressings due to a very small fistula, which later healed.

CHOICE IN METHOD OF SURGICAL TREATMENT

One can never estimate the extent of the damage. It may have reached its limit or it may extend farther after the operation. There are two methods of handling the case. Moynihan suggests the excision I did in this case, and makes the enterostomy openings to use for fluids and food, etc.; others treat the loop as in a Mikulicz operation, and later excise the loop and have the gunbarrel effect, and then anastomose. It is usually inadvisable to anastomose at the time because the patient is in great shock and the added time is an item. Also the edema of the apparently normal bowel is great and makes the operation difficult.

CONCLUSIONS

Occlusion of the mesenteric artery is usually from an embolus followed by thrombosis. In the mesenteric vein it is thrombosis from the start.

The lesions produced are variable, from slight

congestion to marked ulceration and extensive gangrene.

1. The occlusion may be followed by collateral circulation either of a temporary or permanent nature.

2. The occlusion may be followed by cessation of function yet the vitality of the intestine be intact.

3. It may be an infarct with death of a variable amount of the small intestine.

If we have occlusion of a branch and get collateral circulation, we may have slight abdominal pain and distention and these be overshadowed with a greater pathology and the patient survive.

In number two, where the occlusion is in part only, and the function disturbed without the life of the tissues, the symptoms may be those of ulcer or intermittent claudication. These patients in the more severe type have pain incident to food and the tetanic pains like the spasm incident to the anemia. Symptoms of obstruction supervene and the patient may be operated and no obstruction discovered.

In the most severe type, number three, there is definite evidence of obstruction with hemorrhage from the bowels and vomiting, often with blood. These gradually fuse with the clinical findings of peritonitis.

This condition should be regarded as requiring surgery though a few milder cases have had a favorable outcome without surgery.

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DISCUSSION

JOHN HOMER WOOLSEY, M. D. (490 Post Street, San Francisco).—This subject may seem to some as too rare in occurrence to merit the attention and space devoted by the author but, let me emphasize, erroneously so.

I am impressed with three aspects of superior mesenteric thrombosis, all emphasized by Doctor Smith: (1) In making a diagnosis the importance of the history, both antecedent and present; (2) the varying degree of injury and, therefore, variability of symptoms; and (3) the importance of early surgical exploration in this as in any acute, critical and undetermined intra-abdominal complaint.

The antecedent history of an endocarditis, or of any infection with which there might be accompanying emboli; the history of an abdominal injury or the existence of arteriosclerosis are provocative causes subsequent to which arterial mesenteric thrombosis most often occurs. This history of an enteric inflammatory lesion is the one common antecedent of a venous mesenteric thrombosis.

The result of an obliteration of a portion of the arterial supply or the venous return is dependent upon the degree of involvement. Therefore the symptoms and signs will vary as to whether there is only a limited necrosis of mucosa or an extensive infraction of the entire intestinal wall. Undoubtedly people live after a mild degree of mesenteric thrombosis, as they do after acute pancreatitis of a limited degree and in such instances without surgery and, therefore, without a direct view, the diagnosis often remains in doubt.

Surgery today has reached the stage where an intra-abdominal exploration can be done with little to no shock. Early recourse to a direct view of a serious and undetermined intra-abdominal disturbance should be the rule. Mesenteric thrombosis of such a degree as to cause death of a portion of bowel, giving, therefore, symptoms of severe intra-abdominal pain, shock and, as a rule, a marked leukocytosis and later a distended motor function of the intestines, vomiting, and bloody stools, should have prompt intra-abdominal exploration, regardless of whether the diagnosis is, or is not, made. The primary diagnosis in such an instance is "a surgical abdomen" and early diagnosis gives far better returns than malefic procrastination.

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THOMAS O. BURGER, M. D. (1301 Medico-Dental Building, San Diego).—Doctor Smith has added a valuable contribution to the medical literature in this complete discourse on the subject. It is rare, though often enough, to have our attention called to the possibility of such a condition when a diagnosis is not fairly definite.

After taking into consideration the past history, age, and the possibility of emboli, I have made a preoperative diagnosis of occlusion of the vessels in the case of two patients. One of these in mind was inoperable, as proved at autopsy. The second one was opened with the hope that the operative procedure might relieve the condition, but the findings were such that it was hopelessly impossible. In both of these cases there was occlusion of the arterial circulation.

The differentiation between arterial and venous block, I think, is clinically quite difficult, and probably of very little difference insofar as results are concerned.

While thinking of occlusion of the mesentery vessels, we must also think of and consider the possibility of a coronary occlusion in these same types of people. Both these conditions coming from—in the majority of instances—the same source, it is very difficult and almost impossible at times to differentiate from some of the more common catastrophes of the upper abdomen. They are both attended with terrific pain, shock, vomiting, rigidity, and with a leukocytosis in a very short time, also a rise in temperature due to the autolysis of the tissues similar to that of an infection.

It behooves the surgeon to be on the lookout for these conditions from a diagnostic standpoint in order to avoid operating unnecessarily.

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DOCTOR SMITH (Closing).—I appreciate very much the discussion by Dr. John Homer Woolsey in which he emphasizes the etiology of the condition and the importance of early abdominal exploration.

Also the discussion by Dr. Thomas O. Burger in which he also emphasizes the etiology and makes the point that differential diagnosis between arterial and venous block is impossible clinically. To this I fully agree because the treatment is essentially the same.

I only wish to reëmphasize the importance of the careful history, physical examination, and laboratory work. It is quite true that the history, particularly as to cardiovascular disease, is very important, the suddenness of the onset and the acuteness of the pain also are very marked. The urinalysis should always be taken into consideration, because the indican is usually very high due to stasis produced by the paresis of the small intestines. The bloody stools will give a suggestion, and must not be confused with intussusception or enteritis.

I also wish to emphasize that we must limit operation on these patients. They are in extreme shock, ileus is marked, and an anastomosis should not be attempted. It is preferable to do as little as possible, and either to bring the gangrenous loop out of the abdominal wound and fix it there so that at a later date it can be excised and the usual operations for closure performed; or else bring out the loop, suture it into the wound and excise it immediately, leaving two openings, one to relieve the obstruction and the other through which saline may be administered if desired to relieve the toxemia.

TREATMENT OF CANCER—PRESENT DAY RATIONALE*

By ROBERT C. COFFEY, M. D.
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NOTWITHSTANDING the fact that practically every general medical meeting of importance has one or more papers or addresses on cancer and every public health meeting of importance has a discussion of the subject, and every medical institution of research does work on cancer, there has been no notable contribution to our knowledge as to the nature of cancer within a generation. If we would devote our time to systematizing and inculcating our present knowledge of cancer into the minds of the medical profession and the laity instead of following every new theory, we could easily save twice as many cancer patients as we are now saving. Attempts to promulgate new theories as to the nature, cause, and treatment of cancer, and the publication of half-baked theories with no foundation of fact for the reading of the general public so muddles the lay mind that all our attempts to establish the cancer question on a sound scientific basis for the benefit of humanity are largely nullified. So that today the layman is probably no better prepared to decide matters pertaining to cancer than he was twenty-five years ago.

* Read before the general meeting of the California State Medical Association at the fifty-eighth annual session, Coronado, May 6-9, 1929.

PARASITIC THEORY OF CANCER CAUSATION

The parasitic theory is the most harmful of all because it paves the way for the groundless hope of both a preventive and curative serum. The parasitic theory of cancer is not new. It has been brought forward and rejuvenated every time a new advance has been made in bacteriology, beginning with the discovery of the tubercle bacillus. Each time the subject is rejuvenated it is espoused with enthusiasm by certain members of the profession and the laity. Yet, there is no more hope or evidence today that cancer is a parasitic disease than there was when Koch discovered the tubercle bacillus. On the contrary, the evidence is even less convincing. There is strong evidence that cancer is one of the degenerative or terminal diseases; natural processes of maturity and death. This is indicated by the fact that cancer, cardiovascular disease, and kidney disease are all on the increase as far as the general population is concerned, while death from infectious disease is decreasing. The increase of these three diseases run parallel. Why this increase? Is it real or only apparent?

FACTORS IN LIFE EXPECTANCY INCREASE

Forty years ago the average life of the human being born into the world in a civilized country was forty-two years. Today it is fifty-eight years. This increased longevity is chiefly brought about by scientific preventive medicine and hygiene. The mortality from acute infectious diseases, such as diphtheria, malaria, typhoid fever, has been reduced sufficiently to account for most of the increased longevity. Given 100,000 children born today, a much larger per cent of them will die of these three diseases than would have died in 100,000 births forty years ago. But given 100,000 individuals past forty years of age today, there is no evidence to show that a greater per cent of these would die of these three diseases than would have died in 100,000 people past forty years of age forty years ago. This means that there is an enormous increase in the number of people who reach the age of forty, which may be termed middle life, or the turning point at which our body resistance weakens and physical degeneration begins. At this time in life, cancer, cardiovascular disease, and renal disease all begin to develop. The heart valves and blood vessels begin to receive the deposits of lime which replace vital tissues. The kidneys begin to harden and the connective tissue to contract. Normally, new epithelial cells are generated to strengthen and repair areas which are subjected to injury or excessive use. About the age of forty these physical processes begin to wane. Often these repair cells are put into the breach in an immature state and degeneration begins. While the average human life has increased, the maximum longevity has not increased. Man will continue to die. If he escapes accident and infective diseases, he will nevertheless finally die of a degenerative or terminal disease.