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Acute dilatation of the stomach

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ACUTE DILATATION OF THE STOMACH

SENIOR THESIS

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TABLE OF CONTENTS

CHAPTER I	
	page
INTRODUCTION.....	1
DEFINITION AND NOMENCLATURE.....	3
CHAPTER II	
HISTORY.....	6
CHAPTER III	
INCIDENCE.....	15
Sex.....	16
Age.....	17
Occurrence and classification of types.....	19
Predisposing factors.....	26
CHAPTER IV	
ETIOLOGY AND MECHANISM.....	28
Theory of duodenal occlusion.....	28
Theory of gastric paralysis.....	35
Present theory.....	40
Klempner's hypothesis.....	45
CHAPTER V	
PATHOLOGY.....	53
CHAPTER VI	
SYMPTOMS.....	59
CHAPTER VII	
DIAGNOSIS.....	66
Differential diagnosis.....	67
CHAPTER VIII	
COMPLICATIONS.....	69
Cause of death.....	74

TABLE OF CONTENTS

CHAPTER IX

PROGNOSIS.....	page 76
----------------	------------

CHAPTER X

TREATMENT.....	78
----------------	----

CHAPTER XI

CONCLUSIONS.....	89
------------------	----

CHAPTER XII

BIBLIOGRAPHY.....	II-V
-------------------	------

CHAPTER 1

INTRODUCTION

Acute dilatation of the stomach is a rather frequent post operative complication, by some observers it is looked upon as a distinct clinical entity. Although of frequent occurrence it is one of the most commonly overlooked, incorrectly diagnosed and lethal post-operative complications.

It is a just criticism that students of surgery are apt to show a tendency to emphasize surgical technique, and less tendency toward diagnosis, pre-operative or post-operative treatment. There can be no doubt that with the great advances of surgery, as a science and art, there must necessarily be advances in post-operative observation and treatment. Many patients who have been unable to withstand the additional burden placed upon them by surgical treatment might have survived had post-operative treatment been the product of early recognition, good judgement and prompt action.

This condition should not only be of interest to the surgeon, but also to the internist and obstetrician as well. It has been stated that any toxic condition may predispose to its appearance. It has been reported during the course of, or convalescence from severe and

wasting diseases, following childbirth, after falls and injuries, following the removal of body casts, catheterization of the ureters, blows to the abdomen, after errors in diet associated with sudden over loading of the stomach, and in persons with spinal deformity.

Since Rokitansky's (47) suggestion over seventy years ago, that the occlusion of the transverse portion of the duodenum might be the explanation for the production of the disease; Pathologists have in general been skeptical but many surgeons and physiologists have accepted this view. Real progress has been made in the understanding of the constitutional and metabolic changes that make up the late stages of the clinical pictures. However, duodenal obstruction in one form or another has been the dominant idea of many theories advanced, up to the present day, to account for the pathogenesis of the syndrome.

In the preparation of this paper an attempt has been made to review and clarify, as much as possible, the present status of the knowledge of this disease, with especial reference to the many theories advanced to explain the pathogenesis. An attempt has also been made to bring into view its frequent occurrence and the favorable results obtained, providing there is early recognition, and prompt intelligent treatment.

DEFINITION AND NOMENCLATURE

Acute Dilation of the Stomach is a condition in which the stomach rapidly loses its motor power, and is characterized by a sudden and often excessive enlargement of the stomach, the regurgitation of large amounts of fluids, associated with symptoms of a severe toxemia with marked dehydration and collapse.

The term Acute Dilatation of the Stomach is objected to by some authors, especially Harrigan (26), who feels that it is neither complete nor correct, because it fails to denote a dilatation of the duodenum which at times is associated with the former condition. It is of interest to also note that many observers deny this associated finding of dilatation of the duodenum.

A specific cause is not known. Most of the reported cases have followed operations under general anesthesia, and it is probable that the post-operative group comprises from sixty to seventy per cent of all cases. The condition develops from the third day to the third week as a rule, following operation. The occurrence is approximately equal in the sexes and most occur between the ages of ten and forty years, but the disease has been described in an infant of nine months and in a patient eighty-six years of age. From an inspection of the etiology of this disorder, it will be apparent that the

age groups do not give an index of susceptibility but rather of degree of exposure to the factors which may assist in the causation of the condition.

It is the wide variety of these exciting factors, which appear in the literature and the numerous theories made in an attempt to analyse the pathogenesis of this condition that have resulted in a confusing variety of names given to this disorder. This is evident from a glance at some of these synonyms, viz., Acute Dilatation of the Stomach, Acute Gastrectasis, Primary Ectasia of the Stomach, Arterio-Mesenteric Obstruction, Acute Paresis of the Stomach, Gastro-duodenal Dilatation, Gastro-mesenteric Ileus, Post operative Gastric Paralysis, and Acute Gastorrhea or Acute Gastric Succorrhea. The condition is also referred to as Acute Gastro Mesenteric Ileus, although the continental European writers, especially the Germans, describe it under the title of Acute Arterio-mesenteric Compression or Mesenteric Darmverschluss, the later implying duodenal obstruction.

Since the term Acute Dilatation of the Stomach is used by a number of American authors and it coincides with the object of this paper, not to present another new conflicting theory for the causation of the condition but to clarify, the present status of the knowledge of this disease; hence it is chosen as a descriptive title to

express the general information obtained by reviewing
the works of several authors on this subject.

CHAPTER 11

HISTORY

The occurrence of Acute Dilatation of the Stomach is indicated rather strongly in the writings of early observers, judging from the description of symptoms in their writings a great amount of confusion existed and it was not generally recognized.

According to Slocumb (49) one of the first descriptions of the subject is an article written in Latin by Boerneus in 1752, and translated by Dr. J. W. Brannan. He also states Yeats in 1820 described toxic symptoms which he thought were due to compression of the duodenum by the transverse colon, and a case of congenital contraction of the duodenum was reported by Guyot in 1829.

However, the main literature of this condition dates back to 1842, when Rokitansky (47) described a type of Acute Dilatation of the Stomach, due to compression of the "S" loop, or last part of the ileum through the displacement downward of the small intestine. He thought that this type of incarceration was favored by old age, and a long relaxed mesentery. He states, "Dilatation of the Stomach is either spontaneous or is caused by a stenosis. The former represents a uniform size increase and may fill the abdominal cavity. Repeated

repletion, in consequence of a morbid appetite, may give rise to this development, or it may occur as a result of paralysis from concussion traction or dislocation and from hernia. It kills slowly with vomiting, with or without gangrene of the mucous membrane under symptoms of complete paralysis." Although he reported no cases, nor did he state what occurred primarily to cause the mesentery to obstruct the duodenum--nevertheless his work has been of great significance, since many surgeons and physiologists have accepted his views. He was also one of the earliest observers to emphasize and bring to the attention of other physicians its importance as a cause of death.

According to Laffer (33), Miller and Humby published the first case in English literature in 1853, and Bamberger as far back as 1855 called attention to the fact that serious infectious diseases were apt to produce Acute Gastric Dilatation. He also tells of Heschl describing an Ileus due to compression of both the last portions of the duodenum and ileum against the vertebrae, by the mesentery of the small intestine which had been previously displaced into the true pelvis. Also of Erdman in 1858 reporting a case where trauma was the cause, and the patient recovered.

In 1859, Brinton (7) first suggested that Acute Gastric Dilatation was the result of a disturbed innervation

of the stomach wall, with paresis following. This theory has since had the support of many who have written on the subject, although it was for a period of years overshadowed, by the theory of Arteriomesenteric Occlusion of the Duodenum, previously mentioned.

The first good descriptions of this condition were made in 1872 by Hilton Fagge according to Harrigan (26), Laffer (33) and others. This is thought to be one of the outstanding features of the earlier literature, in that the symptoms are detailed, and his article emphasizes the degree of accuracy of observation of these older physicians. He does not mention the etiology and the paper deals mostly with pathology and symptoms. He gathered the few individual case records and notes then existing in text books and transactions of medical and pathological societies. Since some of them were based on a doubtful analysis, they have not been included in many statistical studies. He brought out the point that Acute Dilatation of the Stomach might arise during various diseases. In describing several cases which came under his observation he identified the condition as a clinical entity, altogether distinct from dilatation as a result of stenosis of the pylorus.

Glenard in 1899, as cited after Slocumb (49) stated that Acute Dilatation of the Stomach was brought about by

atony and sinking of the duodenum, causing a narrowing of the duodeno-jejunal flexure which made it difficult for the stomach to empty. He described a physiological closure of the duodenum by the mesentery and superior mesenteric vessels, the function of which was to retain chyme for a longer period in the duodenum. He also pointed out that the empty small intestine when prolapsed into the pelvis exerted a pull of 500 grams on the root of the mesentery.

No cases illustrating the conception that Acute Gastric Dilatation might be due to shutting off of the duodenal lumen by the mesenteric vessels as suggested by Rokitansky (47) were reported until 1891. According to Novak (44), Kundrat in 1891 reported three such cases, all fatal, in a paper with the title "Concerning a Rare Form of Intestinal Incarceration." Post mortem examinations were made in two of these three cases, and both revealed an enormous dilatation of the stomach and duodenum. Also Novak (44) points out, a similar case was reported in 1895, by Schnitzler, who first suggested the postural method of treatment. Schnitzler's work was followed by Albrecht (1), who made a critical study of all the previous literature, and a report of two cases. He also noticed a flattening of the duodenum, between the superior mesenteric artery and the spine. By attaching weights to the mesentery,

he noted that considerable pressure was necessary to force water beyond this constricted area.

Harrigan (26) states, in 1900, Byron Robinson wrote a very comprehensive paper on the subject and endorsed the view of Rokitansky, in a paper based on many dissections. Also in 1900 Mueller (43), considered Acute Dilatation of the Stomach to be the result of lying on the back--or horizontal position. He also raised the question of pre-operative purgation causing a collapse of the intestine, and because of this, a favorable tendency existed for the intestines to sink into the pelvis, aiding the occlusion of the duodenum by stretching the mesentery.

In Campbell Thomson's (52) book written in 1902, it is found he was able to collect 44 cases from the pre-existing literature. Zade, cit. after Kellogg (31) expressed the idea in 1905, in a discussion of post-operative ileus that Dilatation of the Stomach was the most important factor in bringing about compression of the duodenum.

Comner (14) in 1906, made an authoritative study and analysis of the subject. He clearly surveyed the general literature, emphasized and evaluated the important points according to his way of thinking. He does not accept the conclusions of previous investigators, but repeats in detail their experiments, often enlarging upon them from the standpoint of technique. Evidently, he was impressed

by the work of Kelling, who at that time was studying the mechanism of the condition. He repeated and elaborated upon Kelling's work, and altered the technique by the introduction of a water pressure apparatus, to determine the degree of pressure necessary to overcome the obstruction of the duodenum. He also demonstrated upon the cadaver that traction on the mesentery might obstruct the duodenum. He believed the mesentery must be long enough to allow the small intestine to rest on the pelvic floor.

As stated by Novak (44), Finney also wrote on the subject in 1906 and previously in 1902. He was not certain but that Gastro-Mesenteric Ileus, and Acute Dilatation of the Stomach might be one and the same condition.

Laffer (33) 1908, reviewed the literature of Acute Dilatation of the Stomach and Arterio-Mesenteric Ileus, making one of the best analytical and statistical studies up to that time. He recorded a mortality of 63.5 per cent existing at that period.

In 1913 Borchgrevink (5) published an outstanding article on this subject. He reviewed the literature made statistical studies concerning the incidence and prognosis as well as discussing the etiology and treatment. He expressed the idea that the condition of Acute Dilatation of the Stomach paralleled that of Paralytic Ileus.

In 1916 Lee (34) published an interesting paper concerning the post-operative phase of the subject. He reported some of his own cases which occurred during operation and stimulated a great amount of thinking on the subject. A British author Doolin (16) published a paper in 1918. He made a thorough discussion of the subject as well as emphasizing the value of postural treatment in this condition. He made a study of the various types of treatment and the results obtained by each. His statistics showing the high mortality of operative procedure, which greatly discouraged this form of treatment and it has been little used since then.

In 1921, Emil Novak (44) reported some of his cases which occurred during surgical procedure, one of which occurred while the patient was in the Trendelenburg position. These cases with those of Lee (34) have added another type of cases which have developed this condition--those occurring during surgical procedure. He did not agree with the theory of Arteriomesenteric compression and expressed the opinion that the etiology was some form of Gastric Paralysis. Both Swift (51) and Slocumb (49) published papers in 1927. The former consisted of a collection and discussion of those cases occurring during labor and the puerperium while the latter discussed the theory of Arteriomesenteric

Occlusion and stressed its applicability to many cases.

Dragstedt (21) and his co-workers in 1931 did considerable work and published their paper upon "The Pathogenesis of Acute Dilatation of the Stomach" which has had much to do with the formation of our present day concept of the disease. In their discussion of the pathogenesis, they have included some of the points present in the two older theories of the causation of the condition, that of Arterio Mesenteric Occlusion and Gastric Paralysis. They postulated that first there occurred a paralysis of the stomach wall, which aided by Mesenteric Occlusion of the duodenum, even though of slight extent was responsible for the production of the disease. A publication of the clinical and biochemical findings in cases of Acute Dilatation of the Stomach was made by Johnson and Robinowitch (30) a year previously. Kellogg (31) 1931, in his paper has included a summary of the pre-existing ideas as to the causation and treatment of the condition. Ward (54) in 1930 and Wangensteen (53) in 1933, have by their descriptions of their respective suction siphonages apparatuses aided considerably in the treatment of the condition. Curphey and Orr (45) have made a similar contribution along this line.

Klemptner (32) in 1935 expresses some new ideas as to

the possible cause of the condition especially those occurring during operative procedure while Mallory (35) in 1934 has suggested the use of insulin in the treatment of the condition, in an effort to stimulate peristalsis and aid the stomach in regaining its normal tone.

CHAPTER 111

INCIDENCE

The condition of Acute Dilatation of the Stomach shows no particular geographic distribution, nor is it limited to any country. Cases have been reported from the temperate climates, possibly because of more information concerning the condition is found here.

According to Novak (44), in Faggès' original paper in 1873 reference is made in addition to his two personal cases to only a few which had been previously mentioned in the literature, and in 1899 Albrecht (1) had collected eighteen cases. In 1902 Campbell Thomson (52) had gathered forty-four. The study of Conner (14) in 1907 was based on 102 cases. Borchchgrevink (5) 1913, summarizes 144 cases of Acute Dilatation of the stomach. Seven of these were his own and 137 from the literature. Linke in 1914, reviewed 173 cases, 68 of which occurred without operation or narcosis. Doolin (16), in 1918 made a review of the subject and collected 188 cases--to this is added 39 cases by Novak (44) in 1921 bringing the total to 227 cases reported at that time. This seemingly small number of cases collected in the literature, should not be misleading--undoubtedly many cases have occurred without being diagnosed--many others

having been diagnosed, have not been reported, possibly because of lack of interest in the condition. It is true of any condition, that the more interest shown in it, the more apt one is to recognize its beginnings and to become more watchful for its occurrence and course. As Curphey (15) states, "it is doubtful if the medical profession has given this condition quite enough consideration, since in the past five years at least twice as much space in the medical literature has been awarded post-operative Pulmonary Atelectasis, rarely fatal as has been given Acute Gastric Dilatation, a fatal complication if unrecognized and not uncommonly fatal when carefully treated."

According to Morrow (41) every abdominal operation, and most other operations, where there has been either direct or indirect stimulation of the visceral afferent nerves, is followed by varying degrees of gastric relaxation. Since gastric relaxation may be present, it can easily not be recognized and Acute Dilatation of the Stomach may occur unless the physician is watchful for its occurrence and course.

In Borchgrevinks (5) study of 144 cases of Acute Dilatation of the Stomach, which was a continuation of Conner (14) and Laffers (33) studies; of 130 cases where sex was mentioned, 78 patients or 60 per cent were

women, and 52 patients or 40 per cent were men. Thus it is seen that the condition is slightly more prevalent in women than in men.

In 118 cases where age is mentioned the following distribution was found.

1-10 yrs.	1 case	or	0.8 per cent
10-20 yrs.	21 cases	or	17.8 per cent
20-30 yrs.	32 cases	or	27.1 per cent
30-40 yrs.	30 cases	or	25.4 per cent
40-50 yrs.	20 cases	or	16.9 per cent
50-60 yrs.	10 cases	or	8.4 per cent
60-70 yrs.	4 cases	or	3.4 per cent

This is essentially the same as that found by Conner (14), Doolin (16) and others, that three-fourths of the patients acquired their illness between the tenth and fortieth years. However, it may occur at any age. As Novak (44) states, "the youngest patient no doubt was that of Belillios--a suckling infant of 9 months, who was found dead two hours after nursing. The post mortem examination revealed the stomach to be as large as a football." He also tells of the oldest patient recorded, perhaps being that of Moore, the age in this case being 86 years.

Owing to differences in etiology and mechanism all cases of Acute Dilatation of the Stomach may be said to fall into two groups. The first group occurring during or following surgical operations and the stomach is distended by air which is sometimes called--Acute Operative or Acute Post-Operative Dilatation of the Stomach. The

second group may be said to occur in association with certain medical diseases, or during their progress. In the majority of these cases the stomach becomes distended mostly by digestive juices and is sometimes referred to as Acute Secretory Dilatation.

The following table is a summary of the reports of three independent observers, compiled so that one may gain an idea of its frequency of occurrence associated with certain cases. Laffer's, Borchgrevink's, and Linke's cases have been previously referred to and it may be added that Borchgrevink's article dealt mainly with the aspects of the Acute Operative Dilatation.

I Following Operation

	<u>Laffer</u>	<u>Borchgrevink</u>	<u>Linke</u>
following trauma	17	2	10
after laparotomy	9	71	67
after operation on bile tract	15	17	18
after operation on stomach	4	6	12
after operation on female generative organs	12	30	22
after operation on appendix	5	8	0
after operation on intestine	0	5	10
after operation on kidneys	11	5	7
after operation for hernia	4	3	5
after operation on extremities	11	8	9
after extra abdominal operation	0	27	0

II During Progress of Disease:

septic or infectious disease (Typhoid, Pneumonia, etc.)	22	15	18
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	<u>Laffer</u>	<u>Borchgrevink</u>	<u>Linke</u>
emaciating, debilitating disease (Tuberculosis, Carcinoma, etc.)	8	0	22
following errors in diet	0	0	16
spinal deformities	11	0	0

OCCURRENCE AND CLASSIFICATION OF TYPES

If one takes into consideration, the condition of the patients, when Acute Dilatation of the Stomach began, which must be supposed to have more or less affected the development of the dilatation, and group the cases according to the general condition of the patient at the time the dilatation occurred and the circumstances surrounding its onset, it will be found that the cases may be divided into several groups, some of which are fairly well defined.

I The Post-Operative Cases

The post-operative group constitutes the major portion of about 69 per cent of all cases, according to Doolin (16) and Laffer's (33) review of the literature in 1908 which is complete and includes all earlier known cases. Borchgrevink (5) in 1913, found its occurrence in 92 cases out of 138, or occurring as 66.6 per cent in post-operative cases. In this series of 92 cases, 71 were laparotomies and 27 were extra-abdominal operations. Among the laparotomies, operations upon the female genital organs gave the largest and operations upon the gall bladder system the next largest number of occurrences. All of these operations with one exception, were performed

under general anesthesia. The anesthetic administered was in 12 cases chloroform, in 8 ether, in 5 chloroform-ether, in 1 chloroform-ether-oxygen, in 2 chloroform-morphia, in 1 ether-scopolamin-morphia after veronal, in 3 ether-morphia and in 1 ether-morphia-atropin. These are given in order to show, that the beliefs and statements of some observers, that chloroform is especially responsible for the condition, is apparently inconsistent. This is further seen in reviewing Morrows (41) cases, who reported a series of ten cases, including various types of anesthesia, such as local infiltration, ether, ether and gas, spinocain, novocain, and sodium iso-amytal. He believes, Acute Dilatation of the Stomach is not peculiar to any operation or to any anesthetic.

The majority of post-operative dilatation of the stomach occur as an average on the third day following operation, although some have occurred during the first 24 hours, others as late as the sixth or seventh day. It has been observed, that distention of the stomach can occur during operation, and others mention cases occurring as late as the twenty-fourth and thirtieth day following operation. It is probable that these gastric dilatations which appear so late after the effect of the narcosis and operative insult has passed, cannot be correctly

termed post-operative.

II Dilatation of the Stomach which has arisen during the course of an acute illness or its convalescence.

In the review of Borchgrevink (5) fifteen cases out of 138 reported found to belong to this group. Seven were caused by pneumonia, four by typhoid fever and one each by scarletina, acute rheumatic fever, endocarditis, and cholecystitis. In one of these cases, the condition followed taking the first food after three days fasting, and in another patient there was a kyphoscoliosis. The time of appearance was very irregular from the seventh to the twenty-sixth day of the illness.

III Dilatation occurring during chronic illness.

In discussing these in Borchgrevink's (5) group, we find two following pulmonary tuberculosis, one during diabetes and one during sciatica. The majority of these patients were as a rule emaciated and exhausted, in a debility which nearly equalled a post-operative state-- but on the other hand, a few of this group were also reported to be in fairly good physical condition.

IV Dilatation of the Stomach by disorder or deformity of spine.

This grouping includes cases reported of highly developed lumbodorsal scoliosis, spondylitic kyphosis with and without scoliosis as well as other highly

dissimilar and for the most part complicated cases. In one case cited by Borchgrevink (5), there existed a dorso-lumbar scoliosis which hardly left space for a finger between the xiphoid process and the spine. In other cases it seemed hardly reasonable to think that the scoliosis or type of deformity had been the only reason for the dilatation. In fact, in some cases it has been thought that spinal deformity had little or no effect in producing the condition. The reasoning for this was based on the fact that the patient enjoyed ordinary health at the time of occurrence and dilatation arose in direct connection with an overloading of the stomach by food. Five have been reported to have occurred during the application of a plaster jacket.

V. Another group of cases are those in which dilatation is attributed to overloading of the stomach.

Under this heading are included those cases, in which no other explanation could be found for their illness, or where other conditions such as a slight weakness or earlier dyspepsia, were quite insignificant as compared to the single overloading.

According to Borchgrevink's (5) paper we find ten cases comprising this group, six of which died. In three of these the diagnosis was verified both by operation and post mortem examination. This seems to show a severe dilatation can take place after a single

overloading of the stomach. The quickness with which the stomach has been overloaded may play some part with the occurrence of this condition. One case seems to have been due to the quality of food, rather than the quantity of food--supposedly to have been due to a meat poisoning.

VI. Trauma

Laffer (33) and Linke mention seventeen and ten cases, respectively as having occurred following trauma. Dragstedt (21) recently reports one case. Borchgrevink (5) mentions two cases both having followed blows on the abdomen. Both of these patients were sent to the hospital with the diagnosis of ileus. The time elapsing between the injury and the occurrence of the dilatation of the stomach was not mentioned. In both cases dilatation of the stomach was proven both by laparotomy and post mortem.

VII Occurrence during labor and Puerperium.

This condition is rather rare during labor and the puerperium. There is very little written in the text books on obstetrics and the subject has not been mentioned, either as a pathological condition of labor or of the puerperium or as a cause of death after labor.

According to Swift (51) there is a series of 18 cases reported by various authors, although of these, five cannot really be included, as they might have been

due to other causes which were factors in the cases, such as rupture of the uterus, peritonitis, etc.

This leaves 13 cases in this series, and of these one patient died. These thirteen cases might be considered to be a typical series of obstetrical cases, but containing slightly more abnormal conditions than usual. The list includes: spontaneous birth, four cases; low forceps operation, three cases; twins, two cases; breech presentation, one case; eclampsia, one case; transverse position, one case. Swift (51) states, "it was difficult to find any one factor in this series of cases which might predispose to the condition, other than possible operative interference or chloroform anesthesia." Some observers are of the opinion that in cases following normal delivery that Acute Dilatation of the Stomach is due to a sudden releasing of the intra-abdominal pressure, thus favoring a sinking of the intestines and duodenal occlusion.

VIII. Dilatation of the Stomach occurring during operation.

In 1916, Lee (34) added another group, composed of cases in which the dilatation of the stomach occurred on the operating table, while the patient was under the influence of a general anesthetic. He collected and discussed six such cases, including one of his own. In 1921 Novak (44) referred to eleven of these cases, pre-

viously described, and added two which had come under his own observation. Some authors as McIver (37) believe this grouping is characterized by features distinctive enough to warrant its consideration as a definite entity. In each instance, the dilatation has accompanied an operation, not of serious nature; the patients having been under a general anesthetic; the dilatation has been due to a rapid accumulation of gas; the majority of the patients have apparently not shown grave symptoms, in spite of the great distention of the stomach and in most of the cases immediate and permanent reduction of the dilatation has been obtained by passing a stomach tube, allowing the gas to escape. McIver (37) states, "it is natural to conclude that such cases, coming on suddenly and cured by simple means, belong in a different class from the Dilatations of the Stomach which complicates various types of severe illnesses, where the stomach is distended by large quantities of fluids as well as gas, and which are attended by grave symptoms, by a comparatively long course, a marked tendency to recurrences, and a high mortality."

IX. The last group may be termed an idiopathic group, or those cases occurring without an explanation for the dilatation. Borchgrevink (5) mentions two cases, in which the illness commenced with the appearance of ileus, in two apparently healthy subjects, and without

a known cause. He also reports another patient, who was taken ill, after violent laughter.

PREDISPOSING FACTORS

In this discussion, many factors have been mentioned, which might be termed predisposing factors. According to Harrigan (26), Kellogg (31), Starry (50) and others, the following may be predisposing factors.

1. Any toxic condition, the toxicity affecting the nerve centers.
2. Application of plaster jackets for Potts' disease, deformity, injuries in any part of the body, more especially the spinal column and cord, extensive burns and chloroform narcosis.
3. Spinal deformity.
4. Excessive pre-operative purgation.
5. Any operative procedure which rapidly reduces the cubic centimeter dimensions of the abdominal cavity, such as caesarian section and oophorectomy for cyst, without providing for additional support of the cavity.
6. Difficulty in administration of anesthetic, air swallowing, etc.
7. Prolonged dorsal decubitus.
8. Chronic duodenal obstruction.
9. Relaxation of abdominal walls.
10. Ptosis of the viscera, a low position of the third

duodenal segment, bringing it into relation with the prominent lumbar vertebra.

11. Gibson and Wade (24) made a study of the semi-recumbent position or Fowler's position in relation to post-operative dilatation. This has been used as a therapeutic measure in post-operative treatment because many surgeons feel the patients were more comfortable and were less likely to develop pulmonary complications.

In an analysis of 249 cases, 141 of them were treated by the Fowler position and 108 were treated by placing the patient in the flat position. They noticed that in the former method 39 per cent of the patients developed stomach symptoms, while there was an average of 22 per cent in the later method. It was found that the greatest benefit occurred in those patients having had operations upon the biliary system, in which Acute Dilatation of the Stomach was reduced in this series from 43 per cent to 20 per cent. Curiously enough, there was but a small increase in the pulmonary complications, by this method.

CHAPTER IV

ETIOLOGY AND MECHANISM

One of the most interesting problems in connection with this subject concerns the cause and mechanism of this condition. As Doolin (16) states, " a riot of theories have been formed to explain this question." Of these the two which have been most prominent are:

1. the theory of duodenal occlusion by the upper border of the mesentery, with the superior mesenteric vessels, and
2. the theory of paralysis of the muscular wall of the stomach.

There are different opinions as to the exact mechanism by which the duodenum may be compressed by the upper border of the mesentery. According to some the important factor is a sinking into the pelvis of the coils of the intestine, thus causing traction on the mesentery and stretching it--over the horizontal portion of the omentum. Others, again are inclined to believe that the overdistended stomach presses downward on the mesenteric root and thus causes the duodenal occlusion which in turn, precipitates the Acute Dilatation, and thus completing a vicious cycle.

1. Theory of duodenal occlusion

The conception that Acute Gastric Dilatation might be due to shutting off of the duodenal lumen by the mesenteric vessels originated in 1842 by Rokitansky (47).

Others who have supported this view have been Kundrat, Glenard, Albrecht, Robinson, Mueller, Slocumb and many others.

The mechanism of this theory is made more clear by reviewing the anatomy and relationships of the stomach and duodenum.

The duodenum in man is mostly retroperitoneal, and it is firmly fixed by its peritoneal relationship. This fixed portion is further strengthened by the suspensory ligament of Treitz (musculus suspensorius duodeni), which is a musculo-fibrous band extending from the lumbar part of the diaphragm to be inserted into the terminal part of the duodenum and into the mesentery beyond the duodenum. In its course, it passes to the left of the coeliac artery and behind the pancreas. The "C" shaped concavity of the duodenum is occupied by the head of the pancreas with its uncinata process.

The superior mesenteric artery springs from the aorta, and proceeds vertically downward, passing through between the head of the pancreas and its uncinata process to emerge from beneath the transverse mesocolon, after which it crosses anteriorly, the transverse portion of the duodenum and enters between the layers of the mesentery. The vein is located immediately to the right of the artery, and joins the splenic vein behind

the pancreas to form the portal vein.

The lymphatic channels follow the blood vessels in the mesentery, to end in nodes both ventral and dorsal to the pancreas. Those posterior follow the pancreaticoduodenal artery to the hepatic chain, and those anterior follow the superior mesenteric artery to the mesenteric lymph nodes. There is an anastomosis between the lymphatics of the pylorus, duodenum, pancreas and chain along the common duct. The nerve supply of the small intestine is from the sympathetic system, through the superior mesenteric ganglion, which is situated dorsal to the superior mesenteric artery. It also receives fibers from the vagus. The nerves follow the blood vessels and ultimately end in the plexuses of Auerbach and Meissner in the intestinal wall.

Those who support this view believe the relations of the root of the mesentery and the mesenteric vessels predispose the duodenum to danger from compression. Since the small intestine hangs suspended from a vertical, posterior abdominal wall by the mesentery, and must depend for further support upon the anterior abdominal wall and pelvic floor.

Slocumb (49) states, "this arrangement results in a riding over by the root of the mesentery and a compression from the weight of the intestines of the firmly fixed transverse portion of the duodenum. Furthermore,

any considerable pressure at this point would interfere with the circulation, innervation, and lymphatic drainage of the small intestine, since these are all intimately associated in the mesentery at this point. Abnormalities such as peritoneal folds, bands, scoliosis, lordosis and marked visceroptosis might further add to this disadvantage."

These observers infer, the reason for these existing circumstances may be explained by the fact, man has assumed the upright posture. They point out that in the dog, pig and other quadrepal mammals the relations of the peritoneum to the duodenum differ from those in man. The duodenum being a free peritoneal loop in the abdominal cavity of these animals, and is not compressed by the mesentery or the superior mesenteric vessels. The intestines are supported by a mesentery and in addition to a strong horizontal abdominal wall, apparently adapted to the function of supporting their weight.

This view has been replaced more recently, by those who believe in the theory of duodenal occlusion as is found in Bickham's Text book (3)--"Operative Surgery", Miller, Brown and others.

They feel that Acute Dilatation of the Stomach, probably never occurs unless there is a preceding intestinal paresis or atony. This paresis or atony may be produced by surgical shock, traumatic shock,

or shock from any cause. It may also accompany grave conditions such as pneumonia, typhoid and other diseases. They feel that in all of these the pathology is essentially the same, an interference with the nervous mechanism of the gastro-intestinal canal.

It is further explained that with an atonic duodenum lying between the mesentery and the superior mesenteric vessels in front and the resisting lumbar spine behind, considerable pressure is brought upon this part of the duodenum, and a tendency to cause closure of the lumen is developed. A constriction of the lumen of the duodenum would then react upon the lumen of the stomach, and compression of the abdominal wall would also react upon its nerve and blood supply. In a like manner, compression of the superior mesenteric vessels will probably react upon the entire small intestine which would cause a disturbance of both circulation and innervation. Then, with the dilatation of the stomach and its extension downward between the anterior abdominal wall in front and a rigid lumbar spine behind, these structures will become further compressed, and a resulting vicious cycle will be established.

In support of this view are cases of Acute Dilatation of the Stomach occurring in patients with enteroptosis, emaciation from debilitating diseases such as diabetes, typhoid fever, tuberculosis and cases in which the

loops of the ileum have become adherent in the cul-de-sac from localized peritonitis. This view also offers an explanation for the frequency of Acute Dilatation of the Stomach occurring in patients with spinal deformities, such as lordosis, where it is obvious that optimum conditions are present for mesenteric obstruction to the duodenum.

There are many objections to the theory of duodenal occlusion, which up to a few years ago was probably the most widely accepted theory of all. Some of these are:

1. Duodenal Dilatation, although frequently occurs with gastric dilatation is not constant in its occurrence. According to Doolin (16) it occurs in only 25 per cent of the cases and Conner (14) gives the proportion of cases in which a whole or a part of the duodenum is dilated as 38 per cent.

2. There are but a few cases in the literature in which obstruction seems to have been sufficient so as to cause any marked constriction of the duodenum. In Baumler's case, cited by Novak (44) "the post mortem examination disclosed a reddened groove 2 cm. in width, on the upper surface of the bowel, after a release of the constriction.

3. Some observers feel it would be impossible for duodenal occlusion to cause a rapid, sudden Dilatation of the Stomach, as has been seen in a number of cases

which have occurred during operation or the group referred to as reported by Lee (34) and Novak (44).

4. In the cases of very high intestinal obstruction reported Dilatation of the Stomach has not been a common or characteristic symptom. Bloodgood (4) reported four cases of high intestinal obstruction, but in none of these was there more than a slight Dilatation of the Stomach found at operation or post mortem. Borchgrevinck (5) again reports a case of Acute Duodenal Obstruction by a gallstone, emphasizing the fact that in such cases there is an "absence of gastric ectasy and the clinical picture of the Acute Dilatation of the Stomach."

5. It was previously observed that Acute Dilatation of the Stomach is comparatively rare after childbirth, when the intestines might be expected to gravitate into the pelvic cavity after the delivery of the child, which would in this way cause mesenteric constriction of the duodenum.

6. It is also thought, if the duodenal occlusion is an important factor, there would be an indication for gastro-enterosomy. However, as this operation has been done, it carries a very high mortality. Also, if there was a definite mechanical occlusion, it would be hard to explain the fact, that a large porpotion of the patients have recovered by the simple use of the stomach tube without operation or postural treatment.

7. Novak (44) reports two cases of Acute Dilatation of the Stomach occurring after the operation of gastro-enterostomy and infers a number of other similar cases have been reported.

Notwithstanding the many logical objections to this theory--many surgeons are of the opinion at present that this may explain the mechanism of the dilatation. It will be observed, in the discussion of the present day theory, that many parts of this theory, exert a favorable influence in the minds of various authors and have been incorporated into the present conception of the mechanism.

II. Theory of Gastric Paralysis.

This theory cannot be as clearly and specifically presented as the previous theory, largely due to the fact of conflicting reports in experimental evidence and data. Although this theory for a number of years was over shadowed by the theory of arterio mesenteric occlusion, it has since had the support of many who have written on the subject.

The occurrence of Acute Dilatation of the Stomach, during the course of an abdominal operation, in the Trendelenburg position, as reported by Novak (44) in 1921, strongly suggests that the mechanism is probably due to some form of nervous reflex.

This theory is not new however, Novak(44) himself credits Brinton (7) in 1859 of being the first to suggest the theory of gastric paralysis--according to Brinton (5) "Acute Dilatation of the stomach is the result of a disturbed innervation of the stomach wall, with paresis following."

In the description of this theory, it is necessary to mention some of the facts in connection with the physiology of the nerves and motor activity of the stomach, as determined by experimental investigation, in so far as they bear on the question under discussion.

Post-operative intestinal paralysis has been studied by Cannon and Murphy (10) who noticed the following to occur: The intestines were etherized, from one-half to one and one-half hours--then after exposure of the intestines to the air, with unusual cooling, the motor function of the stomach was not greatly impaired. However, handling of the intestine caused an indefinite delay in their motor power; even manipulation under warm salt solution checked gastric peristalsis for three hours. Fingering gently in the air caused greater retardation of movement, and after rough handling in air, no food was noticed to pass the pylorus for four hours, and then it moved slowly.

According to Kellogg (31) Kelling has described a valve-like closure of the cardia, which is opened by

contraction of muscle fibers, extending from the esophagus into the fundus. Kelling believed that weakening of these fibers or an increase in the intragastric pressure could close this valve-like mechanism, which associated with an obstruction to the duodenum, could produce the condition of Acute Dilatation of the Stomach.

As discussed previously in this paper, the vagus and splanchnics are the extrinsic nerves to the stomach. Brown, McSwinnney and Wade (8) have made a thorough investigation concerning the effect of sympathetic stimulation upon the stomach. In previous literature, both inhibitory and augmentor effects have been described as to the effects of stimulation of the sympathetic system upon the movements of the stomach--causing much disagreement. They point out the fact that in preliminary experiments which were made, it was shown that the anesthetic had an influence upon the effect of sympathetic stimulation, and that motor or inhibitor responses could be obtained, according to the anesthetic used. They used anesthetized cats and both spinal and decerebrate cats and dogs in their experiments, and the effects of stimulation, both upon the body and pylorus of the stomach were observed according to their technique. Their experiments appeared to show:

- (a) the sympathetic innervation, after complete severance from the rest of the stomach, was still retained at the pylorus.
- (b) fibers were in the sympathetic chain which conveyed inhibitory impulses to the stomach as a whole.
- (c) that motor fibers to the body of the stomach were also present.

The effects on the stomach, seemed to depend upon the type of stimulation of the splanchnic nerves employed. It was noticed that a stimulation at a frequency of one per second caused contraction of the body of the stomach-- and stimulation with a strong, tetanizing current brought about relaxation. The movement of the cats' pyloric antrum were inhibited by all forms of stimulation-- and the antrum of the dog, although usually inhibited, occasionally showed an augmentor response to stimulation of the sympathetics.

In 1895 Carrion and Hallion, cited by Doolin (16) demonstrated that section of the vagus nerves in dogs, resulted in Dilatation of the Stomach. A demonstration of the effect of Vagus section in the human was observed and reported by Hartwell (27). He noticed Acute Dilatation of the Stomach following the accidental severance of the vagus nerve during a difficult thyroid-ectomy. However, Dragstedt and Dragstedt (17) deny

these observations. In their paper in 1921, they state, "although the operation has been performed a great number of times in the study of various problems, a section of both vagus nerves above the diaphragm, or a combined section of both vagus nerves and splanchnic nerves in dogs, has never in the experience of many observers produced a dilated stomach."

In the reasoning of this theory, by the observers who support it, as Novak (44), Doolin (16) and others, claim it is no more remarkable that abdominal injury or manipulation may reflexly produce gastric paralysis, than that a pithed frog's heart may be made to stop in diastole by light tapping on the frog's abdominal wall with the handle of a scalpel. Similarly, also they reason, that a sudden local paralysis of the uterine wall is occasionally observed during curettage, and the relaxation may become quite marked. They feel perhaps that the afferent impulse might have its origin elsewhere than in the abdominal cavity for instance in those cases following injuries to the extremities. They also feel that in the nonsurgical cases, the mechanism would seem to be a purely local one. They are not certain as to whether the reflex has to do chiefly with an inhibition of the vagus or a stimulation of the splanchnic nerves.

In Novak's (44) summary of this theory in 1921, the

following statement is significant. "All in all, however, it may be said that the weight of evidence is rather over-whelmingly in favor of the paralytic theory of post-operative dilatation of the stomach, that this theory, more than any other, explains the varied immediate etiology of the condition, and that it alone would seem to explain those rapidly occurring dilatations observed during operations."

PRESENT THEORY

More recent work has somewhat clarified the idea concerning the mechanism of Acute Dilatation of the Stomach. The conception of a gastric paralysis in the usual sense, through interference with its motor innervation, and apart from reflex or toxic depression of tonus, is thought by most observers to be not quite true. The works of Goltz and Gaskell, cited by Dragstedt (21), Carlson and Luckhard (11), Alvarez (2), and many others, indicate the intestinal tract to possess a peripheral automaticity, similar to that of the heart or amphibian lung, and a section of its extrinsic nerves, will not produce a paralysis any more than a section of the vagus or sympathetic branches to the heart, will result in paralysis of the cardiac musculature.

Carlson and Luckhardt (11) have shown that it is possible to obtain visceral reflexes, from the stimulation of either somatic or visceral sensory nerves. Although

both motor and inhibitory fibers are present in the vagus nerve, the predominant effect of the vagus on the tonus of the mammalian cardiac and pyloric sphincters is inhibitory, and upon the stomach proper, is motor. It is thought the opposite situation is obtained in the effect of the sympathetic fibers from the coeliac plexus. Although both motor and inhibitory efferent fibers are also present the main effect of the sympathetic fibers on the tonus of the cardiac and pyloric sphincters is motor, and on the body of the stomach, it is inhibitory.

In 1931, L. R. Dragstedt (21) reports he has observed, a relaxation of the stomach with contraction of the cardia and pylorus, following direct stimulation of the peripheral splanchnic nerves in cats under light ether anesthesia, or reflexly through stimulation of many of the abdominal viscera and of sensory fibers in the sciatic nerve. Relaxation, but to a lesser degree has been observed after section of the vagi.

The degree of relaxation which is seen, in some cases of Acute Dilatation of the Stomach in the human, cannot be readily reproduced in laboratory animals. This being explained in various ways--some think this may be due to a species difference, or it may be possible that some depression in the peripheral gastric motor mechanism may be necessary. Since there is some

experimental evidence as well as clinical observation reported, mainly by Payer, cited after Doolin (16), that chloroform or ether anesthesia depresses the gastric tonus and this may be an important contributory factor.

The nervous pathways involved, in the visceral reflexes are indicated in the diagram which has been taken from Dragstedt (21), et al.

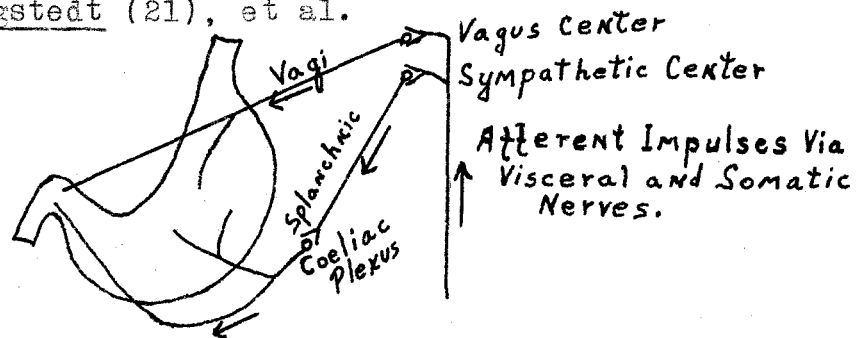


Fig: 1. Diagram illustrating the pathways of nervous impulses resulting in reflex gastric dilatation from the stimulation of visceral or somatic sensory nerves. There are, of course, intercalated neurones connecting the sensory and motor nerves here shown. Reflex stimulation of the sympathetic center is accompanied by a simultaneous inhibition of the antagonist or vagus center, an example of so called reciprocal innervation.

It was pointed out previously in this paper that most of the cases of Acute Dilatation of the Stomach, have occurred after laparotomy, and, of these, operations

upon the gall bladder and female generative tract seem more prone than others to cause the condition. The occurrence of Acute Dilatation of the Stomach following injuries and extra--abdominal operations, such as those on the extremities and face, have also been mentioned. It is believed that profound stimulation of somatic as well as visceral sensory nerves, may bring about reflex inhibition of the stomach. According to Dragstedt, Montgomery, Ellis, and Matthews, (21) in their recent paper, "evidence is thought to be adequate, to show how trauma incident to laparotomy, or operative injury to any part of the body, or child birth, might cause marked inhibition of the gastric tonus, through reflex inhibition of the vagus and stimulation of the sympathetic nerves to the stomach."

It is further believed that Dilatation of the Stomach may be produced, as a result of swallowed air, and accumulating secretions of the stomach and duodenum.

The above authors, have reported a case in which they believe a secondary occlusion of the duodenum has been demonstrated. The patient, a robust male, had received a severe blow to the abdomen, and the attachment of the root of the mesentery to a large portion of the small intestine had been severed. A jejunostomy was performed. On the third day following the operation, examination revealed, a dilated stomach containing about

1400 cc. of characteristic fluid. Aspiration of the fluid greatly relieved the patient, and shortly the drainage from the jejunostomy again became profuse. It was believed, the accumulating fluid in the dilating stomach had evidently produced a secondary obstruction to the duodenum and prevented the escape of pancreatic and duodenal secretions into the jejunum. The withdrawal of the material in the stomach, apparently had relieved the duodenal obstruction and allowed the escape of digestive secretions from the jejunostomy.

Whether the occlusion of the duodenum was due to a direct pressure of the stomach with its large amount of fluid or to mesenteric compression could not be determined. It is thought that the degree of obstruction in these cases is probably not great, since it is found in many post mortem examinations, the obstruction can be easily overcome, by the examining finger.

Nevertheless, in the presence of a profound reflex inhibition of the motor functions of the stomach, and possibly of the duodenum, together with the overdistention produced by gas and accumulating secretions, even the slightest obstruction may be of the greatest importance, in that the atonic stomach and duodenum are not able to propel their secretions into the lower intestine.

KLEMPNER'S HYPOTHESIS

The theories previously described are not endorsed by several authorities and new explanations are being offered, for the causation of the condition of Acute Dilatation of the stomach, as in times past. One of the latest, is that of Klemptner (32) brought forward in 1935. It has been given the term Klemptner's Hypothesis, in this paper, for the sake of clarification and to lessen confusion. It is included in this paper, for the sake of completeness and because in general it contains some of the ideas of several recent authors on the subject.

As previously mentioned in this paper due to the fact, there are differences in etiology and mechanism, we can classify all cases of Acute Dilatation of the Stomach into two groups. The first group occurs during surgical operations and the stomach is distended by air, it is designated Acute Operative Dilatation. The second group occurs in the post-operative state or in association with certain medical diseases, and the stomach becomes distended by digestive juices, and this has been termed Acute Secretory Dilatation. A discussion of each will be made, including the recent ideas of Klemptner (32) and others.

1. Acute Operative Dilatation

There are some symptoms which are not uncommonly

mentioned in the histories of cases recorded in literature. Some of these are: the patients take the anesthetic poorly, their color is often bad and they struggle a great deal before the anesthetic has taken effect.

Alvarez (2) in 1928, recorded findings, while doing animal experimentations, with their abdomen open, in which he could often notice a peculiar type of powerful respiratory movement, which rapidly filled their stomachs with air.

As noticed above, the common factors in several of the cases reported, was a disturbed respiratory mechanism. Normal breathing is purposeful and efficient, and the proper muscles are brought into play, the rate and depth of respiration having been adjusted to best utilize the oxygen in the air. Klemptner (32) points out, that the introduction of unusual, difficult environmental conditions upsets this balance of the respiratory mechanism.

It is thought in this hypothesis, that the patient who breathes in air mixed with vapors of ether or chloroform feels like choking, unless the anesthesia is deep. He then struggles, respiration becomes difficult, and disorganized, extraneous muscles are brought into play, and whatever else may happen in some cases the muscles of deglutition begin to work synchronously with

the expiratory muscles and air is forced downward into the stomach, until the pressure there is in excess to the atmosphere.

The swallowing of air then distends the stomach and brings on the syndrome, which is more than a reflex act, in fact it might be termed a biological reaction--the expression of a struggle for adjustment to abnormal environmental conditions.

2. Acute Secretory Dilatation.

According to this hypothesis, two factors are essential for the production of this type of Acute Dilatation of the Stomach. They are: (a) reverse peristalsis and (b) suppression of the act of vomiting.

(A) Reverse Peristalsis.

The contents of the intestinal tract are thought to move caudad, because of the arrangement of graded forces along the wall of the intestinal tract. This is the theory of intestinal gradients of Alvarez (2). The wall of the upper part of the tract is thought to have a higher tonicity, rhythmicity and metabolic rate as compared to that of the lower bowel, and the stomach acts as the "chief motor" of the system.

As a result of pathological conditions a rearrangement of areas of high and low pressure, may be allowed to take place. The lower bowel may develop areas of increased tonicity through congestion and irritation and

the stomach may become atonic. The intestinal gradient then may become flattened or reversed, carrying intestinal contents to the stomach by peristaltic waves. Reversed peristalsis is then expressed clinically, by nausea and vomiting.

The reversal of gradient is thought to be, mechanically directly related, to the atony of the upper intestinal tract--as a result of the reflex inhibition of the vagal branches or the stimulation of the splanchnic nerves.

The anti-peristaltic waves carry fluids from the intestines to the stomach. If for some reason, the stomach fails to empty itself, by vomiting, it becomes distended and the distention reacts upon the motor and secretory functions of the digestive tract.

The low tonus of the upper intestinal tract, is thought to be an important factor for the reversal of the intestinal gradient, which is still further depressed by the distention of the stomach: the reversed intestinal gradient is steepened and the stomach is turned into an aspirating apparatus for the digestive juices and the contents of the small bowel. In other words, the reverse peristalsis causes the stomach to be distended and the distention intensifies the reverse peristalsis thereby creating a vicious cycle.

By aspirating the contents of the stomach, the

stomach is given a chance to contract and to regain its normal tonus, thereby reestablishing the normal intestinal gradient and ending the syndrome.

In support of these ideas some pathological conditions are mentioned with associated reverse peristalsis. Some of these are: exogenous poisons, ether, chloroform, nitrous oxide; various forms of toxemia, as uremia, acidosis, eclampsia, endocrine disorders; infectuous fever diseases of the intestinal tract; migraine, tabetic crises, pregnancy, shock and others.

Klemptner (32) believes a striking coincidence is present, since most all of these pathological conditions are associated with reverse peristalsis--and most of them have been stated to be etiological factors in the production of Acute Dilatation of the Stomach.

In regard to the theory of duodenal obstruction by compression, he maintains that definite duodenal obstruction does not produce the syndrome, either in the laboratory or clinically. He believes the essential factor to be the irritation, produced by compression or pinching of the intestinal wall and not the occlusion. This pinching or compression, causes an area of increased tonicity to be produced, the upper intestinal gradient to be reversed and the digestive juices are backed up into the stomach, with all of the associated phenomena which follows.

In review of Dragstedts' (21) reported case, previously cited in this paper and in which Dragstedt (21) made the conclusions; that aspiration of the stomach relieved the compression of the duodenum, but no definite explanation was offered for the stopping of the drainage from the fistula primarily and why the stomach became distended. In reviewing this same case Klemptner (32) reached the conclusion that the stoppage of the drainage and the Dilatation of the Stomach were due to reverse peristalsis. He believes, reversed peristalsis to be of common occurrence in the presence of a jejunal fistula of this kind. It was further thought that, as a result of aspiration of the stomach, it was allowed to regain its normal tone with the reestablishing of normal peristalsis, thus giving relief to the patient. Klemptner (32) states, "the alternating change in direction of the intestinal gradient was responsible both for the production, as well as for relief of the syndrome."

(B) Suppression of Vomiting.

Since vomiting is a complex act and being made up of voluntary and involuntary components, it is thought that as long as the stomach can empty itself, it will not become distended. During the act of vomiting, the cardia and fundus relax, and contraction of the abdominal muscles, the diaphragm and the antral portion of the stomach, forces the contents into the esophagus,

and here a sudden expiratory movement carries the vomitus over the rima glottis. The act of vomiting is thought by most physiologists to be a protective measure.

Klemptner (32) believes as long as the stomach can empty itself by vomiting, it will not become distended. If vomiting is suppressed or inefficient and some degree of atony is present, the stomach would be unprotected against the rush of fluids, carried there by reverse peristaltic waves and dilatation takes place.

Some of the causes for suppression of vomiting are thought to be:

1. extreme debility
2. conditions where vomiting is painful, as pneumonia and operative wounds of upper abdomen.
3. a toxic patient, with depression of vomiting center.
4. suppression of vomiting by use of palliative drugs, in the course of medical treatment.

Since opiates are given for pain in the post-operative state, it is thought this may be a factor. These combined with the effect of the anesthetic or of the pathology present may account for some of the reverse peristalsis present in the post-operative state.

Although this hypothesis is recent, and sufficient time has not elapsed for the reports of authorities,

either critical or otherwise, nevertheless some of the ideas contained will probably play an important part in the solution of the problem.

Many minor theories have only been touched upon, and some have been omitted in this discussion, because they have long ago been discarded by the various authorities. Only those, which have had followers, over long periods of time and exert influence upon our modern concepts of the causation of the condition have been discussed. However, it is interesting to review these theories in regard to the etiology and mechanism for this condition--in which there has been as yet, no specific cause assigned. One new theory is rapidly replacing another in an effort to give an explanation which will cover the field. It is doubtful if Acute Dilatation of the Stomach can be ascribed to one single cause but rather to a number of causes, each of which may play its own important role, in sequential order in the production of Acute Dilatation of the Stomach.

CHAPTER V

PATHOLOGY

The study of the gross pathology of Acute Dilatation of the Stomach, is as yet, in some respects incomplete. More information would be desirable concerning the question, of equal involvement of the anterior and posterior walls of the stomach, and the relation of the transverse colon to the dilated stomach, needs some clarification. The histology as found in various reports is also rather indefinite.

However, the stomach is found to be always more or less distended. The distention in some cases is comparatively slight and presents itself principally, by the globular shape of the stomach. In other cases, the stomach is quite distended, and extends downward into the pelvis, and often to the symphysis pubis. Under these circumstances Borchgrevinck (5) quotes Fenger's description, who states, "the stomach looks like a huge, bent arm with the elbow near the pelvis." Between these extremes many variations are found. The excessively distended wall of the stomach is usually described as being quite thin, and in some the contents may be observed through the thin wall. Its color is red or bluish, and with lesser degrees of distention, it may be gray or gray-blue.

In the mucous membrane, regular extravasations of blood are reported to be found and often superficial erosions, which may explain the hemorrhagic nature of the vomitus.

Henry (28) describes degenerative changes in the mucosa of the duodenum associated with this condition which takes place early, and undergoes necrosis. Conner (14) in a study of sixty nine autopsies, concluded there was an associated dilatation of a part or whole of the duodenum. In thirty eight cases in which the duodenum was dilated, the dilatation stopped abruptly near the lower end of the duodenum, where it passes behind the root of the mesentery, causing a definite obstruction. In eight cases there was dilatation of the duodenum, but no compression was found.

Some authors as Bloodgood (4), Albrecht (1), Bäumler (cit. by Borchgrevinck)(5), and others, have described a furrow or constriction of the duodenum, as being a rose colored stripe on the serosa, and corresponding to it a necrosis in the mucous membrane, which has been ascribed to arteriomesenteric--compression. Since this has been reported in but a few cases, the concensus of opinion is that it can hardly be taken, to be indicative of arterio-mesenteric compression. In his discussion, Borchgrevinck (5)

dismisses this evidence by the statement "for the remark, that we find only, that for which we are looking can also be applied to in a post mortem examination."

Concerning the small intestines, the literature contains little information. In most instances, when it is mentioned they have generally been described as collapsed and lying in the pelvis. Sometimes it has been noted that there has been a long mesentery associated with these findings.

The microscopical picture, is that of an extremely stretched, thin, stomach wall with small hemorrhagic areas in the mucous membrane, and as reported by Henry (23) evidence of superficial erosions and associated evidence of degeneration. Dragstedt et al (21) denies there is evidence to show that the distention has interfered with the blood supply to the gastric wall and describes the mucous membrane as being pale. Kellogg (31) describes the gastric wall as being edematous.

The contents of the stomach have been reported to be chiefly gas and a thin greenish, greenish-brown, or brownish-black liquid, which is odorless and practically neutral in reaction.

The experiments of Dragstedt and Dragstedt (17) in 1922 suggests, that in conditions similar to those found existing in Acute Dilatation of the Stomach,

There occurs an absorption of toxic substances, from the duodenum which have a pronounced secretagogue action. They theorize, that duodenal obstruction secondary to the gastric dilatation, results in the development of an increased tension in the obstructed duodenum. This increased intraviseral pressure allows the absorption of toxic materials which are otherwise excluded by the selecting absorbing function of the intestinal mucosa. These toxic substances which have been formed, then cause a pronounced stimulation of the gastric, pancreatic and duodenal secretions. This factor may play a role in increasing the out flow of digestive fluids in all conditions accompanied by obstruction in the intestines.

The enormous quantity of fluid that is regurgitated, or may be aspirated from the dilated stomach is one of the most striking features of the disease. Most all observers have commented on the excessive amount of fluid and most are agreed that the fluid is a composite of gastric juice, pancreatic juice, bile, and the secretions of the duodenal mucosa.

It has been difficult to obtain accurate information regarding the total volume of the various digestive secretions secreted in twenty-four hours, under normal conditions in man. Rowntree (48) in 1922, did a great amount of work on this subject and the following table

is taken from his article.

<u>Secretions</u>	<u>cc. per 24 hrs.</u>
Saliva	1500
Gastric juice	2000-3000
Bile	300-500
Pancreatic juice	500-800
Succus entericus	3000

Recent work indicates that these estimates may be slightly low. Nevertheless, most authorities on the subject feel that the enormous quantity present is far in excess to the normal volume secreted. They believe this hypersecretion is a result of proteolytic action, in which histamin--like bodies are formed in the duodenum, and these powerfully stimulate the secretion of the digestive glands.

The origin of the gas found in the stomach has also been the subject of speculation. McIver (37) in his studies of the disease has done some work along these lines.

Fermentation was thought to be an important factor in many cases with a slow onset. In those cases with a rapid onset as reported by Lee (34) and Novak (44) the possibility of this being a factor is rather small because of the suddenness of the distention.

The possibility that gas might be due to a secretion of carbon dioxide from the blood stream, was suggested to Woodyatt and Graham (cit. after McIver (37) by Schierbock's theory that carbon dioxide was secreted into the stomach under normal conditions. After a

series of experiments, they concluded that such a secretory activity might be responsible, at least in part, for the gas present in Acute Dilatation of the Stomach. However more recent studies by McIver, Redfield and Benedict (38) have shown that the movement of carbon dioxide into and out of the stomach, seems to proceed according to the physical laws governing the diffusion of gases, and there is no reason to suppose, the gastric mucosa could assume a secretory activity of this nature.

Diffusion from the blood stream, has also been studied as a possible origin of the gas present. However it is thought by most observers, that the amount of gas entering the stomach by diffusion, would be too small to play an important part, especially in those cases in which there has been a rapid Dilatation of the Stomach.

Atmospheric air is held by more recent authorities as being the possible origin of the gas. It having been aspirated during the struggle in difficult anesthesia, aeorphagics etc., It would seem that this might be a logical explanation for the gas in those cases in which there is a rapid distention.

CHAPTER VI

SYMPTOMS

As a rule, in post-operative cases, the symptoms do not present themselves until the third day after operation, but even prior to then certain consistent factors of importance may be noticed. At first, the patient is apathetic and comfortable, the mind is clear, the pulse may be normal or slightly increased in rate and small in volume. The blood pressure is not increased and the temperature may be normal or sub-normal, unless the condition complicates a febrile state.

The patient may begin to show some apprehension, and a constant distress in the upper abdomen may be present. There will be, with the use of enemata, passage of flatus from the bowel, without relief of the upper abdominal distress. Examination at this time, will show no fluctuation, but a tympanic note may be observed over a large area of the upper abdomen. Passage of the stomach tube at this time will give immediate relief--and it will be found in most cases that the primary distention is due, not to a fluid content of the stomach but almost entirely to gas. Only mild, if any, toxic symptoms will be present.

In cases, when these first observations have not been made, and in those cases which progress, after some judicious treatment has been instigated, a striking

picture presents itself. The mode of onset, as well as the time of appearance, or the development and degree of the major symptoms, such as nausea, vomiting, pain, abdominal distention, toxemia, dehydration and collapse may be variable. In the less severe cases pain, nausea, epigastric fullness, regurgitation and moderate distention are usually observed. Seemingly the better the patient beforehand, the symptoms are more sharply defined, when Dilatation of the Stomach makes its onset. The illness, is thus seen in a more clear form after a less serious operation. In a case of medium severity, the dilatation will as a rule show itself by the patient complaining of pain and distention of the upper abdomen together with increasing thirst. He will then begin to vomit, without relief following. The vomiting occurs at short intervals, and when once started, becomes one of the most prominent features of the disease. A distention of the stomach occurs rapidly. From the beginning it includes the epigastrium, by degrees it extends downwards, past the umbilicus and can perhaps reach the lower part of the abdomen. As a rule the distention is largest in the left side of the abdomen, while the right takes little if any part.

Upon examination of the abdomen one will find tenderness, which begins in the epigastrium, and extends

downward with the increasing Dilatation of the Stomach. It is of greatest intensity, during the beginning of the dilatation--the excessively distended stomach often is less tender and at times no tenderness is present. The outlines of the stomach are for the most part indistinguishable. There are cases reported, in which an excessively distended stomach, has not had the slightest influence on the shape of the abdomen.

In the ordinary course of the disease, splashing sounds may be elicited by percussion, or shaking of the abdomen will reveal a large collection of fluid or gas, by hearing a "succussion splash" in the stethoscope. If the distended stomach is displaced upward, succussion sounds may be produced by the heart beats, and occur simultaneously with the movements of the heart. According to some European authors quoted by Borchgrevinck (5) as Baumler, Braun and Seidel, Haberer, Heine-Ewald, Herrick, Lehmann and Stuart, and Bloodgood (4) in the United States, the outlines of the distended stomach are indicated by a symptom, which gives the highly interesting evidence, the stomach is not paralyzed, namely, that of visible peristalsis--although this is denied by many.

If a stomach tube is introduced, gas and fluid will be expelled. If one is in doubt as to the seat

of the distention, this should allay his fears when the distended stomach collapses.

If the condition goes on the regurgitation increases, the pulse becomes fast and small, the urine is scanty, the patient becomes prostrated and toxic with symptoms of collapse, characterized by a cold, clammy skin, cold extremities, some degree of cyanosis with rapid and shallow respirations, anxious expression and the patient passes into delirium or coma.

The chief symptoms of this condition are vomiting, abdominal distention and collapse and symptoms of secondary rank are pain and tenderness over the distended stomach, thirst and scanty urine.

Vomiting is present in about ninety percent of the cases, and usually is the first to attract attention. Vomiting may be present and then disappear for hours or even days, only to return later. It has been described as "regurgitant", "persistent", "uncontrollable", "profuse", and "incessant". The vomitus is usually a dark greenish, flocculent fluid and may be odorless, or foul smelling, but not fecal in type. The quantity is great. Chemical examination of the vomitus in a number of instances show bile to usually be present. Both a hyperchlorhydria and a achlorhydria have been found as well as a normal content of hydrochloric acid. Hydrogen sulphide has been found in some cases as well as yeast

cells, bacterium coli, together with staphylococci and streptococci.

Distention is usually present, it may affect mostly the epigastrium, the left side of the abdomen or involve the entire abdomen. Pain is present in about twenty-five per cent of the cases, and may be severe. Abdominal tenderness is usually present in most all cases.

Collapse is generally present, it usually occurs early, especially in the severe cases. It is thought to be due to over-stretching of the stomach walls, loss of body fluids, interference with respiration and heart action and to the toxemia produced.

Thirst is one of the most common symptoms, and at times it becomes quite severe and agonizing to the patient.

Intestinal action seems to be quite independent of the Dilatation of the Stomach. Flatus and movements of the bowels have often both ceased, while in other cases there has been an accompanying diarrhea.

The temperature unless influenced by an associated condition is usually normal or sub-normal, while the pulse becomes fast and small.

Occasional reported symptoms have been hiccough, marked dilatation of the superficial abdominal veins and edema of both legs.

The scanty urine and urinary changes seem to bear

a direct relation to the severity of the toxicity which is thought to have its origin, as a result of the activity of the proteolytic bacteria and the presence of so called toxic bodies which exert a secretagogue action causing the enormous amount of fluid in the stomach.

The urinary changes may show: indican, albumin, granular casts, cylindroids, high specific gravity and a decreased output.

The blood changes are also important. With the progressive dehydration, there is an increase in hemoglobin and in the number of red cells. The leucocytes may vary in number from 7,000 to 14,000.

The blood chemistry, has been found to show a decrease in the plasma chlorides, an increase in the carbon dioxide carrying capacity of the blood and increase in urea and non-protein nitrogen. Johnson and Rabinowitch (30) have recently made a study of the clinical and bio-chemical findings in Acute Dilatation of the Stomach. They attribute the accumulation of non-protein nitrogen in the blood not to be due to impairment of kidney function but to tissue destruction. They have also found the increase in blood urea nitrogen to parallel the clinical picture and the creatinine to be normal or practically so. They add another finding, which may be present, that of a mild,

though definite hyperglycemia which is also thought to be the result of necrosis of the mucosa.

CHAPTER VII

DIAGNOSIS

The early diagnosis of this condition is very important, in regard to the prognosis of Acute Dilatation of the Stomach. It should not, as a rule, present unusual difficulties, although this condition is often over looked, merely because the physician does not have this condition in mind.

In many cases, the first glance at the patient, will note a brownish green or black stain at the angles of the mouth, where the regurgitated fluid has soiled the skin. There is an anxious facies and the patient is apprehensive. The pulse is increased in rate and smaller in volume, while no marked change in the temperature has taken place.

Physical examination, reveals the abdomen to be distended, first occurring in the epigastrium, and later involving the left side or entire abdomen. Pain with abdominal distress and tenderness will probably be present in most cases and the succussion splash can be elicited in the greater majority of the patients.

These findings, with the rather characteristic symptoms, the large amount of gas and fluid revealed by passing the stomach tube with relief of the patient by lavage, together with the urinary and blood changes should make the diagnosis rather quickly, with no

unusual difficulty. X-ray examination has proven to be very useful in those cases in which there has been some doubt to the diagnosis, when the above findings present a confusing picture.

DIFFERENTIAL DIAGNOSIS

This condition may simulate post-operative ileus, intestinal obstruction, and peritonitis.

The differentiation will be suggested by the characteristics of Acute Dilatation of the Stomach as, the absence of fever, the frequent and copious vomiting of the regurgitant type, the absence of marked abdominal tenderness or rigidity, absence of marked leucocytosis and most important, the striking temporary disappearance of the distention after the passage of the stomach tube, with a large amount of brown colored fluid escaping.

In both peritonitis and intestinal obstruction, the abdominal enlargement is more diffuse and rounded, and is not relieved a great deal by gastric lavage. The amount of vomitus, and of the material recovered by lavage, is also commonly much less. In addition there is usually more or less fever and leucocytosis in peritonitis. In post-operative ileus, the onset may not occur as early as does that of Acute Dilatation of the Stomach, pain is more severe, and vomiting is not such a major symptom.

If attention is first directed to the urinary

changes, it may be confused with uremia, the differentiation should not be difficult, from bearing in mind the above statements. The blood changes will differentiate it from acidosis.

Occasionally reported conditions, which may cause some confusion in diagnosis have been:

- (a) Large gallstone, causing obstruction, in the duodenojejunal region.
- (b) Congenital or hypertrophic stenosis at the pylorus.
- (c) Retroperitoneal hernia.
- (d) Post-operative vomiting.
- (e) Appendicitis.
- (f) Hematoma of the head of the pancreas.
- (g) Acute hemorrhagic pancreatitis.
- (h) Diaphragmatic hernia.
- (i) Kidney colic, ovarian cyst and gastric crises of Tabes.

These conditions offer little difficulty to diagnosis, the chief reliance can be placed on the passage of the stomach tube with relief, the characteristic fluid, aided by the history and the points of difference in the symptoms which may be present, as well as laboratory findings.

CHAPTER VIII
COMPLICATIONS

The chief factors involved in the production of the complications of this condition, may be ascribed to an alteration in the blood chemistry of the body. Under normal conditions, the gastric and pancreatic juices, are secreted and poured into the upper part of the alimentary tract and are more or less completely absorbed in the lower part of the intestine. The water and inorganic salts, which are the principal constituents of these secretions, are not apparently absorbed in the stomach, duodenum, or upper jejunum--but in the lower jejunum, ileum, and colon, they are more readily absorbed.

In order, then for reabsorption of the gastric and pancreatic juices, motor activities of the intestines are necessary, to carry them into the ileum and colon. Interference with this transport will result in the loss of various constituents of these secretions to the body, either through vomiting or accumulation in the lumen, of the non-absorbing portions of the tract. The loss of gastric juice has been offered as an explanation for the hypochloremia, alkalosis and dehydration which exists. The conception that harm might result from the failure of reabsorption of gastric and pancreatic juices, has largely come about through experimental work upon the

pathogenesis of high intestinal obstruction by McCallum (36), and others.

Other experiments, demonstrating the fatal effect of the uncomplicated total loss of gastric juice in the experimental animal have been reported in some detail by Dragstedt and Ellis (19) in 1930. In their work, the continuous total loss of gastric secretion under conditions as nearly normal as possible, and in which there was no obstruction in any part of the alimentary tract was secured. It was found that, although the animals were given adequate food and water, they became weak rapidly, depressed, losing from one-third to one-half of their body weight, and died in from five to eight days. With the above symptoms, characteristic changes in the blood chemistry were noted. There was a fall in the concentration of chloride, an increase in the carbon dioxide combining power of the plasma, a shift in the hydrogen ion concentration toward the alkaline side and a late increase in the non-protein and urea nitrogen. The changes in the blood chemistry were roughly proportional to the severity of the symptoms, and both were without question due to the removal or loss of the gastric juice.

The important elements lost in the gastric juice are contained in physiological salt solution. They found, by the administration of adequate amounts intravenously, the changes in the blood chemistry could be prevented and

the life of the animal prolonged. The inability of water alone, to fill those purposes, indicates that it is the ions of sodium and chlorine, that are of the greatest importance. These are lost in the secretions, since they cannot be absorbed in the upper intestine, and they are not taken to the absorbing portion, in this condition, to be returned to the blood stream, for life to continue.

Of the other digestive juices, the effects of their loss has also been studied. Dragstedt, Dragstedt, McClintock and Chase (18), in 1918, showed that total loss of duodenal juice, did not show outstanding harmful effects. In many cases of biliary fistula, observed in various clinics and hospitals, has shown the total escape of bile does not produce acute symptoms.

Elman and McCaughan (22) in their work in 1927, were the first to demonstrate clearly that the continuous loss of the major portion of the pancreatic secretion, would cause death in a short time. Dragstedt, Ellis and Montgomery (20) in 1924, secured data on the effect of the total loss of pancreatic juice by a more refined technique. They found the animals to recover from the operative procedure, but shortly after they were noticed to lose weight steadily, gradually become weak and depressed with death occurring in the course of five or six days.

In many respects, the blood chemistry showed changes

which were opposite to that described, following the loss of gastric juice. There was a decrease in the concentration of fixed base, to a less extent a decrease in the chloride, a marked decrease in the carbon dioxide combining power of the plasma, and a shift in the hydrogen ion concentration toward the acid side. The dehydration accounted for the loss in body weight but the blood volume remained normal even during the terminal stages. The administration of pancreatic juice by mouth was found to prolong life and correct the altered blood chemistry, showing death resulted in the continuous removal of pancreatic juice. It was also found, that by daily administration of from 2,000 to 3,000 cubic centimeters of physiological salt solution by intravenous injection, life could be prolonged and the altered blood chemistry could be, in part corrected. This also seemed to give proof, that the loss of the elements, sodium and chlorine are responsible for the fatal outcome, when they are continuously removed.

It is then evident, that the property of the gastric and pancreatic glands, to remove inorganic elements such as sodium and chlorine, from the blood plasma makes it necessary that these, and other portions of the secretions, must pass into the absorbing portion of the intestine, in order that they can be returned to the blood stream and avert these complications found in

this condition.

The occasional symptoms of tetany, which have been observed in patients with Acute Dilatation of the Stomach, are thought to be due to the alteration in the chemistry of the blood because of this failure of reabsorption of the excreted digestive juices. An extensive alkalosis exists if there is a marked loss of the acid gastric juice--and tetany is observed in the marked alkalosis which exists, as a result of forced breathing. Dragstedt, Montgomery, Ellis and Matthews (21) report they have been unable to reproduce this finding in dogs, in which extreme alkalosis was reproduced by the draining gastric fistula. However, it is to be noted that in severe alkalosis produced by hyperpnea and in which tetany occurs, there is also a reduction in the carbon dioxide tension in the blood. In the severe alkalosis following the loss of gastric juice the hydrogen ion concentration of the plasma may be low, but the carbon dioxide tension and carbon dioxide content are above normal. It may be that a combination of hyperpnea and loss of fixed acid may produce the alteration in blood chemistry necessary for tetany.

McLaughlin and Levering (39) have made studies concerning the effects of the increased intragastric pressure upon thoracic and abdominal arterial and Venous pressures. In their experiments on dogs, they

have produced the picture of Acute Dilatation of the Stomach as seen in clinical cases. However, since the progressive increase in pressure, in the animal were induced in a relatively short period of time, and without the loss of blood volume which may occur with a more insidious onset, it was concluded that the terminal effects on the cardio-vascular system of the human might be even greater than their experiments disclosed. They observed, whenever the intragastric pressure was increased, the pressure in the inferior vena cava rose promptly and with decreasing intragastric pressure, there was also a decrease in the pressure of the inferior vena cava.

Slight increases in the intragastric tension caused no changes in the superior vena caval pressure, however, if it were over 30 millimeters (mercury) there was a slight rise in the pressure. They also observed, as the intragastric pressure was increased there was usually, a fall in the pressure as recorded from the femoral artery. There resulted also, an obstruction to the portal blood flow and evidence was gained, which pointed to a tamponade effect on the abdominal aorta from the distended stomach.

CAUSE OF DEATH

It is the conclusion of most authors, that the cause of death is a chemical one. The failure of reabsorption of

the gastric and pancreatic juice with the accompanying dehydration, toxemia and loss of sodium and chlorine ions, can account for the lethal outcome, in many cases in which the pathologist has been unable to demonstrate a secondary occlusion of the duodenum. It is thought to be probable in many cases, that atony alone has led to the failure of reabsorption of digestive juices, because they are not taken to the absorbing portions of the intestinal canal. It is believed, however, when secondary occlusion is found to be present, even though it be of a very slight extent, it may form a successful barrier, to the feeble peristalsis which might be present in the proximal portion of the duodenum.

Another contribution to this phase of the subject has been made by Brams, Katz and Kohn (6) since they have observed, that after the release of marked abdominal distention in dogs, which had persisted for some time, the fall in the arterial pressure was as much as 40 millimeters (mercury). They caution, that such a drop in a feeble patient, might result in death, if the tamponade effect of the abdominal arterial and venous system is suddenly reduced. The readjustment of the circulation depending upon the rapidity of the restoration of the normal factors controlling it.

CHAPTER IX

PROGNOSIS

Many authors in discussing the prognosis of this condition, believe the literature does not give a correct expression for the prognosis of the illness, in that many less severe and cured cases are not included, because they are overlooked. There might be some doubt however, as to the correctness of this statement, because an Acute Dilatation of the Stomach has very little, if any disposition to spontaneous cure. There is a very wide range of variability of the onset of this condition, and varying degrees of severity; it is true also that its course is protracted in many cases. Many cases have been treated with the stomach tube, received fluids and general supportive measures, and have died in less than a day. In several cases the dilatation has reappeared after the first food or drink, and in some it has continued for some time, even after the severe symptoms have disappeared. Borchgrevink (5) tells of the x-ray examination of one patient, which revealed the stomach to reach three centimeters below the umbilicus, a month after the onset.

As stated before, an Acutely Dilated Stomach shows little or no tendency to spontaneous cure, and practically all of the unrecognized and untreated cases, have a fatal issue. However, of recent years, the medical profession

has become aware of the possibilities of this condition, and diagnosis has been made with more accuracy, and prompt treatment instituted, which has improved the prognosis to a marked degree. This will become evident, by viewing the mortality rates of three different periods.

<u>Year</u>	<u>Author</u>	<u>Mortality</u>
1907	Conner	72%
1908	Laffer	63%
1913	Borchgrevink	54%

No recent author has made an exact computation of the present mortality rate, however, the general concensus of opinion is that it is possibly in the neighborhood of 7%, or slightly lower, in all cases of Acute Dilatation of the Stomach.

Doolin (16) has given a more true perception of the patients chances, by analyzing the mortality rate as recorded, by the various methods of treatment as seen in the table below.

<u>No. of cases</u>	<u>Type of treatment</u>	<u>Deaths</u>	<u>Percent Mortality</u>
31	medically	29	93%
29	operation	21	72%
54	lavage	25	50%
30	postural	2	7%

These figures are not only significant in viewing the mortality rate as a whole, but also in the choice of treatment to select.

CHAPTER X

TREATMENT

The treatment is not the least interesting phase in this peculiar, and formerly nearly always fatal, illness. In the rapid development therapy has undergone during the past fifty years, restoration to health has taken place in a great number of cases, which formerly would have been more or less hopeless. Some of the greatest triumphs have been achieved by operative surgery. With Acute Dilatation of the Stomach, however, this has been obliged to admit defeat, and on the other hand, results which can compare with some of surgery's best have been attained by the simplest means.

Before entering upon the treatment of Acute Dilatation of the Stomach after it is once established, it might be well to consider some points in prophylaxis as stressed by Kellogg (31) and others.

1. Before operation, avoid strong catharsis and prolonged fasting.
2. During operation, avoid unnecessary exposure and manipulation of the intestines.
3. After operation, avoid prolonged dorsal and Fowlers' position as much as possible, and keep the patient on the right side or on the abdomen, if not contraindicated.
4. If the small intestines are in the pelvis, at time of operation use the Trendelenburg position, if possible.
5. Foods and fluids by mouth, should be given in small quantities at first, to avoid distention.
6. With the onset of digestive discomfort, belching, nausea and regurgitation, aspirate the stomach contents.
7. Empty the stomach early and repeat the procedure frequently if there is a tendency for fluid in the stomach, to collect rapidly.

After the condition is once established, the indications are:

1. To keep the stomach empty.
2. To restore the body fluids and combat alkalosis.
3. To relieve possible mesteneric compression.
4. Stimulate peristalsis.

In regard to the first of these procedures many methods have been used to keep the stomach empty, all of which have been met with a favorable degree of success. An energetic and continual evacuation of the stomach has become one of the principal methods of treatment in cases of Acute Dilatation of the Stomach. This method also seems to conform with good reasoning and rationality in that it relieves a distended, hypersecreting stomach. The more soon and often this is done, the better is the result.

If the evacuations take place only after the stomach is distended to its maximum and the patient's strength has weakened and almost exhausted, aspiration of the contents of the stomach will, in many of these cases fail to obtain the desired result. An early and complete emptying of the stomach is desirable.

One of the more early methods used to accomplish this result was by gastric lavage, and much has been written concerning the value of gastric lavage both before and after operations. Most authorities agree that this procedure has saved many lives under certain circumstances, such as in the earlier stages of Acute

Dilatation of the Stomach. The lavage of the stomach should be continued until the fluid has returned clear, is the view expressed by Moynihan (42), Finney (23), Horsely (29), Bickham (3), Slocumb (49), and others.

Recently, some objections have been brought forward in regard to the use of gastric lavage. In a number of threatened and existing cases of Acute Dilatation of the Stomach, on the surgical service of United Israel-- Zion Hospital from 1922-1927 Wolfson and Kaufmann (55), employed the simple procedure of tube passage and gastric lavage was omitted entirely. The results, demonstrated to these authors that passage of the stomach tube is superior to the more difficult task of lavaging the stomach. They point out, the preparation for the procedure is more simple and easy, there is less discomfort to the patient, and the danger of aspiration pneumonia is thought to be decreased. It also has the advantage, in that it can be used at more frequent intervals, and the already lowered chloride content of the blood, will not be further reduced. They feel, that with the introduction of large quantities of fluid into the stomach, there is a washing out of the hydrochloric acid from the cells of the gastric mucosa, which might be saved other-wise.

In more recent years, it has become the custom to

use the stomach tube for aspiration purposes rather than gastric lavage. This has proven to be effective in many cases. The stomach tube should be of moderate size with a velvet like finish. The openings should be placed laterally near the tip and their combined caliber should exceed that of the lumen. The margins should be smooth and rounded and the tip, hollow and soft.

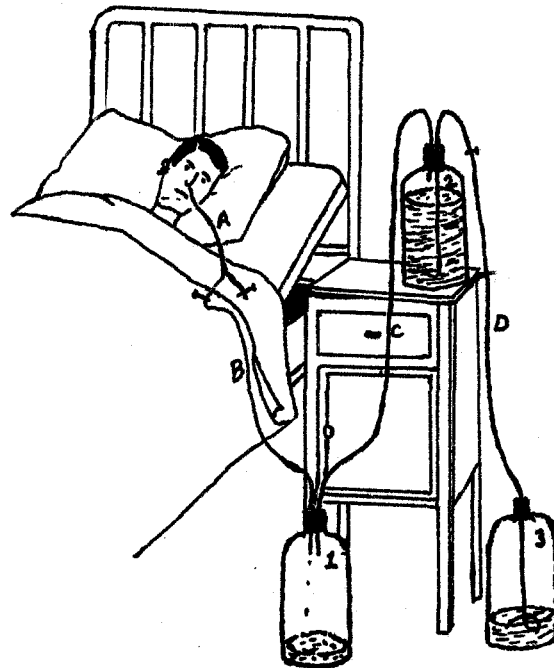
In some cases, difficulty is encountered, in passing the tube. The greatly distended stomach, contains gas and liquid, and the latter will, providing the patient is lying in bed, more or less mould the thin and yielding posterior stomach wall, to the excavation of the posterior abdominal wall. It is not always easy in these cases, to bring the tip of the tube, under the surface of the liquid, making it necessary for one to try tubing with the patient in various positions.

It is necessary to repeat this procedure often enough to prevent the reaccumulation of toxic products, making it necessary to repeat the passage of the tube in the majority of cases every three or four hours day and night. The disadvantage to these procedures, is obvious, and in cases where frequent aspiration is required, it is desirable to leave the tube in place with continuous drainage. The stomach tube has another disadvantage in that it does not adequately empty the

stomach of both liquid and gas.

The most effective procedure is one which utilizes continued drainage, by supplying a source of constant suction which forcibly empties the stomach until its tone can be regained. When this method is in use, the stomach can be constantly washed by allowing the patient to drink water as desired, which also is of aid in combating the prominent symptom of thirst. In addition to the mechanical emptying of the stomach, careful attention must be given to the water and chemical balance of the body.

Several types of suction apparatus for this purpose have been described such as that of Ward (54) and Wangensteen (53) respectively. Recently, however, Orr and Curphey (45) have devised a similar apparatus, which seemingly offers more advantages in ease of operation than that of the above authors and is equally effective, as will be seen in the accompanying diagram.



Suction apparatus used for gastric and duodenal decompression and lavage. A. Levin tube with Y-tube attached for ready cleansing with syringe. B. Rubber tube leading from patient to waste bottle no. 1. D. Rubber tube connecting water supply bottle with overflow water bottle no. 3. Bottles no. 2 and no. 3 are interchangeable to avoid new supply of water when no. 2 runs low. Tube D. Kept filled with water with lower end immersed in water of bottle no. 3 to maintain continuous suction.

This instrument has the advantages, in that it requires no additional bed side apparatus, other than the bed side table; it segregates the gastric contents, giving examination and an estimation of the gastric secretions which are lost. It can be assembled with little skill in a short period of time, and at little

expense. It is simple to operate, requiring little attention which is extremely important in a busy hospital.

The suction procedure, may be began or ended at will, until improvement is shown, by a diminution in the quantity of secretion, a less offensive odor and it becomes lighter in color as well as the general improvement in the symptoms of the patient.

In addition to emptying the contents of the stomach and keeping the stomach empty until its normal tone can be regained, it is also necessary to give careful attention to the water and chemical balance of the body. Dehydration may be extreme in some cases and the loss of chlorides from the stomach may result in marked hypochloremia. Water and sodium chloride may be given daily, as normal saline solution, by three different methods--venoclysis, hypodermoclysis, or proctoclysis. The quantity of chloride to be given, can be controlled by frequent blood chloride estimations. As a supportive measure glucose is generally given in a five per cent solution with the normal saline solution, by the venoclysis method. Usually three thousand to four thousand cubic centimeters of normal saline solution are given daily in the vein. In addition to restoring body fluids these procedures will also combat

alkalosis.

Some authors as Kellogg (31), Slocumb (49), and others, feel as though mesenteric compression, has a great part in the causation of the condition, and treatment to relieve this should be instigated. In many cases this method of treatment, termed postural treatment by most authors, has given very good results, when the stomach tube has done no good. The adoption of the prone position, according to Doolin (16) was first recommended by Schnitzler, of Vienna, in 1895. It has been used in many cases since then, reducing the mortality from sixty per cent to seven per cent according to his figures.

This is a very simple procedure. An abdominal binder may be applied with a pad above the pubis and the foot of the bed elevated. In suitable cases, the knee chest position may be assumed with the abdomen supported by pillows. In other cases, the patient may be placed upon his right side with the foot of the bed elevated which seems to afford a great deal of relief.

Condon (13) recommends, reversing the position of the bed, and floating the small intestines upwards, by filling the abdominal cavity with Ringer's solution introduced by needle puncture of the abdomen. The addition of glucose solution seems to prolong the effects of this method of treatment, by delaying

absorption. Slocumb (49) in speaking of this method makes the warning, "with any marked degree of gastric dilatation, there would be great danger of the needle entering the stomach. An accident of this kind was reported in the British Journal of Surgery, 1921-1922."

In the earlier days, the surgeon felt obliged to operate the patient, when the treatment with the stomach tube did little or no good. However, it is not encouraging to view the high mortality resulting from such surgical treatment. Doolin (16) has made a statistical analysis of this phase of the subject, and of the cases subjected to operative treatment, five varying surgical procedures have been carried out with the following results.

1. Gastrotomy with evacuation of the contents of the dilated stomach was done on six patients, with five deaths.
2. Gastrostomy was used in seven cases, with six deaths.
3. Gastro-enterostomy was employed seven times with four deaths.
4. Jejunostomy was fatal in the only case in which it was tried.
5. Exploratory laparotomy was performed on eight cases and the dilated stomach on recognition, was emptied either by tube or by manual compression. This procedure was followed by five deaths.

It is thus seen, in thirty-one cases, which have been operated upon, only eight have recovered. This procedure has been abandoned by most surgeons of recent years because of the high mortality.

A large amount of medication and remedies has been given in an effort to stimulate peristalsis and to aid the stomach and intestines to regain their normal tone.

Physostigmine salicylate has been given, and is useful in some cases, but it has the objection of being a circulatory depressant. Pituitrin has been used by many and found to be slightly less effective.

Kellogg (31) advises the use of an enema, containing six per cent salt solution, which he has found to be effective in stimulating peristalsis. He believes there is little indication for other remedies as atropine, warm compresses, turpentine stupes or strychnine.

Since the beginning of the use of insulin, it has been known that a full dose would cause hunger sensations. In 1913, Carlson (11) showed that sensations of hunger were associated with increased gastric tonus and motility. In 1924, it was first shown by Butalao and Carlson (9) that insulin increased gastric motility and injections of dextrose solutions, seem to abolish it. La Barre, cited by Mallory (35) showed that the action was through the vagus nerves, by demonstrating in cross circulation experiments that when the hypoglycemic blood of one animal was passed through the cerebrum of a second, the gastric function was increased in the second animal although all connections with the cerebrum were

severed, except the vagus nerves.

Mallory (35) with these facts in mind, suggested the use of insulin in this condition, in addition to emptying the stomach and combating dehydration by venoclysis. He reports a case in which insulin was used with good results. The patient was a male age 53 years who had an Acute Dilatation of the Stomach following a cholecystectomy. This was treated by aspiration of the stomach contents, with intravenous injections of normal saline solution with glucose and with ten units of insulin. The two following days, the patient received two hundred cubic centimeters of orange juice and insulin five units, after each meal and intermediate feedings. No further vomiting occurred and the patient made an uneventful recovery.

In conclusion Mallory (35) states, "complete repeated evacuation of the stomach and prevention of dehydration remain essential in the treatment of Acute Post-operative Gastric Dilatation. Insulin would seem to be a valuable physiological remedy both in the prevention and treatment."

CHAPTER XI

CONCLUSIONS

In the preparation of this paper an attempt has been made to discuss all phases of the subject of Acute Dilatation of the Stomach, and in the presentation to clarify as much as possible the knowledge of this condition as found, existing to date in the literature.

An interest in this subject was aroused by a study of two cases in 1935. One having followed an appendectomy, the other followed an operation for mechanical intestinal obstruction. Although an early diagnosis was made and prompt treatment instituted in each of these cases, the first had a fatal outcome. Since this condition is a cause of death in numerous medical and surgical conditions, there is no doubt many other cases of the same nature are presenting such a problem, so as to require physicians and surgeons to be constantly watching for their occurrence.

From the facts presented, much work is yet to be done, to determine the true etiology and mechanism of this condition as well as the pathology. Much progress has been made in the study of the complications and treatment. It is gratifying to view the lowered mortality rate. No doubt the better prognosis may be attributed to--an earlier diagnosis and an improvement in pre-operative and post-operative treatment in surgical patients.

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