presence of an upper G.I. lesion. The x-ray diagnosis of leiomyosarcoma is seldom made preoperatively. While it is much too early to claim cure in either of these cases, the fact remains that one year and one month following operation when one of our patients died from another cause, no evidence of residual tumour was found. In the other case one year following operation the patient is living and well with no evidence of recurrence. It would appear that it would be difficult to make the diagnosis of leiomyosarcoma of the stomach with any degree of certainty in a large percentage of cases. However, the diagnosis can be made preoperatively by the roentgenologist with careful study in some cases as exemplified by Case 1. The treatment of this lesion is surgical. A wide resection of the involved portion of the stomach offers the best chance of the patient being well in the future.

SUMMARY

1. Two cases of leiomyosarcoma of the stomach are reported with a brief review of the literature.

2. In the consideration of sudden massive upper gastro-intestinal hæmorrhage which fails to respond in any way to conservative therapy, this lesion should not be overlooked.

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HICCUP

A ten year review of anatomy, etiology, and treatment

LESTER SAMUELS, M.B., Ch.B., M.D.,(Sheffield),† *Kew Gardens, N.Y.*

PAST MEDICAL LITERATURE provides few references to the etiology and treatment of this interesting malady. A study of efforts made to define and classify its causative factors terminates in the conclusion that most of the findings and attendant conclusions cannot be justified in the light of modern advance. Charles Mayo[1] in one of the early published articles on the etiology of hiccup frankly admits the dearth of information on the subject. His classification, based upon the established anatomy and physiology of the day, remains the one clearly cut effort extant. Excellent though it is, it fails to satisfy the demands of present day investigation. Soviet Union researchers,[2] basing their conclusions upon a misconception of the mechanics of hiccup, fail to show any appreciable advance in the knowledge of fundamental causation. Hamelin,[3] in his description of hiccup as a rhythmical, clonic contraction of the diaphragm gives far too broad an explanation to be of assistance in building an understandable classification of the malady. Remaining publications on the subject concern themselves mainly with modes of treatment, with scant effort at enquiry into etiology or pathology.

*From the Surgical Services of the Memorial Hospital of Queens, Jamaica, New York.
†Former Chief of Surgical Service, Veterans' Hospital, Alexandria, La., Moore and Johnston General Hospitals, U.S. Army.

ANATOMY

In view of findings in the actual mechanics of the hiccup spasm, a study of all anatomical structures involved must form a basis for any true pattern of its etiology. The modern concept of phrenic and sympathetic nerve anatomy becomes an integral part of the consideration of the diaphragm as a whole. Kuntz,[4] in his able work in the sympathetic field, describes hiccup as an involuntary reflex, respiratory in nature, and purely automatic. We believe the respiratory manifestations of the spasm to be secondary to initial muscular contraction. He also quotes Regelsberger's comments on the part played by the sympathetic system. Regelsberger,[5] however, makes little mention of sympathetic influence in hiccup, and disregards other sources of motor innervation to the muscular origins of the diaphragm. Kuntz,[4] also states that the existence of a special centre in the brain which mediates the hiccup phenomenon, is improbable. Though, with present knowledge this may be true, experience in this series leads us to believe that the combined action of the respiratory and phrenic centres, together with the hypothalamic sympathetic centres, may actually perform the work of a specific centre. Cases mentioned in case histories below tend to strengthen this belief

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in that the causative factor in the attacks occurred directly in brain tissue.

It is unfortunate that so important an anatomical structure as the diaphragm does not appear to have attracted any sustained attention in the overall picture of modern anatomic advance. Through the years the basic description of this muscle follows a set pattern in edition after edition of standard text books. Goetz,[6] Kuntz,[4] Smithwick and White,[7] Mandl *et al.*,[8] have in their recent publications provided an incentive for further intensive research in this direction. Present descriptions lead one to believe that the diaphragm is a predominantly tendinous organ. Repeated dissections show a preponderance of muscular tissue in almost 70% of its wide circumferential bases of origin. The central tendon

particular, in view of the powerful part they play in diaphragmatic movement, must be considered: (1) The area of interdigitation between the lower costal bundles and those of the transversus abdominis muscles. These areas are innervated by branches of the anterior branches of the lower three thoracic nerves. Scant evidence can be found of phrenico-sympathetic nerve supply to these bundles. (2) The crura. These, without doubt form the most powerful origins of the diaphragm. Only the medial portions of these elongated, thick muscular bundles, and the supporting medial and lateral arcuate ligaments are entirely fibrous. Both Cunningham and Callander[10] make mention of the fact that the crura may be innervated by the lower thoracic nerves. We have found that whereas phrenic

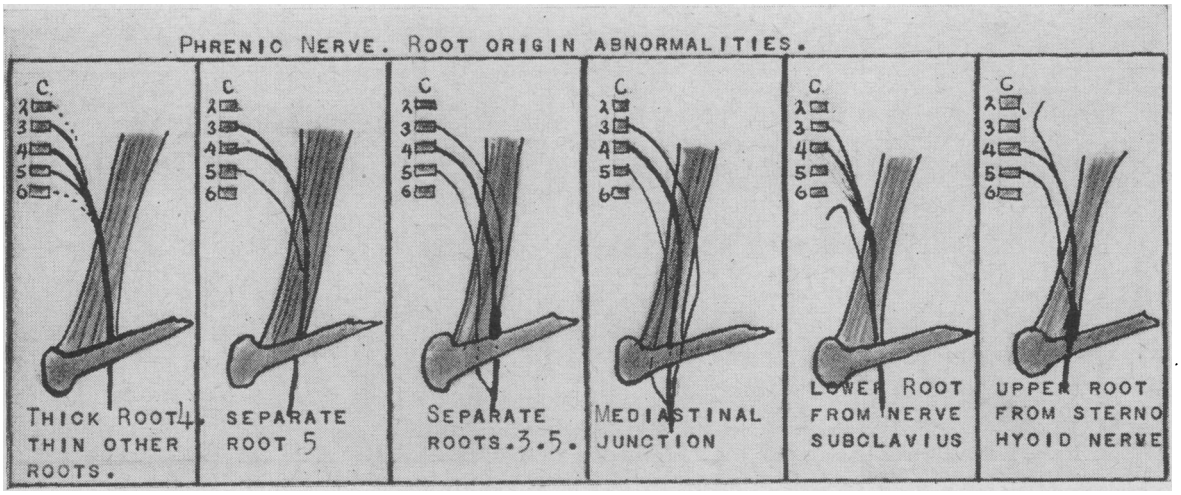

Fig. 1

—so-called—is not a tendon and is not central. Musculo-fibrous bundles are found throughout its entire extent. It is situated posterior to the dome centre and always a little lateral to the mid line. The fibres of this so-called tendon do not converge into a central core but run through its whole area without any set pattern. Surgeons experienced in diaphragmatic surgery have long recognized the difficulty of obtaining a straight line cleavage of this part of the diaphragm. Consideration of the tremendously wide areas of origin, extending from the ziphoid process anteriorly, through the whole extent of the lower thoracic cage to the upper three lumbar vertebræ impresses the dissector with the large amount of purely muscular tissue found.

The actual innervation of these peripheral areas assumes importance in the study of mechanics of the hiccup spasm. Two areas in

and sympathetic supply to the crura is very limited, branches from the lower two thoracic and upper two lumbar nerves are very numerous. Thus we can no longer consider the phrenic nerve as the paramount innervating force of the diaphragm as a whole. These differences in the innervation of the central and outlying portions of the diaphragm form a definite basis of explanation of the varying types of hiccup contractures that will be outlined later.

In order to facilitate etiological classification, the phrenic nerve and its sympathetic connections will be considered in two sections:

Cervical and mediastinal section.—A newer concept of phrenic nerve anatomy readily supplies the answer to many of the problems of the etiology, mechanics, and treatment of hiccup spasm (Fig. 1). The bare statement found in text book anatomies, that the nerve arises from cervical 3, 4, and 5 is only partially correct. Cunningham,[9] conscious of the fact that abnormalities are frequent, states: "The nerve is derived mainly from the fourth cervical, reinforced by roots through from the

third cervical (either directly or through the nerve to the sterno-hyoid), and fifth, either directly or through the nerve to the subclavius". He further states that abnormalities in the formation of the nerve are found in 20 to 30% of dissections. Callander,[10] agrees as to the existence of an accessory phrenic nerve in the same percentage. Thorek,[11] in his admirable surgical treatise describes communications between the phrenic and the vagus, superior and inferior cervical sympathetic ganglia, the spinal accessory and hypoglossal nerves. He agrees with the previous authors as to the percentage of accessory phrenics found, and states that one can be reasonably certain that all connections with the main trunk are severed if 15 cm. of the trunk are removed. Although this is not recommended or thought necessary at any time, it is a far cry indeed from simple phrenic crush as a means of stopping all phrenic innervation (See operative technique below).

Further study amply demonstrates these and other abnormalities. Branches may arise from cervical 2 and 6. The branch from cervical 5 often remains as a separate fine nerve which does not rejoin the main trunk for varying distances extending to the superior mediastinum. It is usually found medial to the main trunk and must not be confused with the sympathetic chain. A thick root from cervical 4 and a fine root from cervical 3 may be found separate to any level in the neck. The author has twice encountered this abnormality during operative procedure. Though the possibilities of failure of simple phrenic crush must be apparent with the known percentage of abnormal phrenic trunk formation, this does not by any means, complete the picture.

In addition to references made by Thorek above, Kirgis and Kuntz,[12] Siwe,[13] Heinbecker and Bishop, *et al.*[14] furnish a detailed account of a series of intercommunicating fibres between the phrenic, vagus and sympathetic chains in the cervical region. Funaoka,[15] states that these communicating strands are found to be more numerous on the right side, and that on many occasions he has failed to find vagal communication fibres on the left. With reference to this finding it is curious to note that the great majority of hiccup spasms are unilateral and confined to the left diaphragm. Though it is not now possible to discuss the presence of sensory fibres in the vagus and sympathetic trunk, such an eventuality must be considered in the overall picture of etiology and treatment. Some of the above authors mention the possibility of some of these communicating fibres leaving the phrenic nerve for the sympathetic and vagus trunks and rejoining the parent nerve at a lower level. The name cervical phrenic circle has been applied to the above anatomic findings.

Abdominal section.—Cunningham[9] states that the phrenic nerve is the main motor nerve of the diaphragm, and that there may be some innervation from the lower thoracic nerves. He makes note of the fact that upon reaching the diaphragm the phrenic divides into many branches, the majority of which terminate in the subperitoneal areas. The structure and complexity of what is now known as the diaphragmatic plexus receives scant attention from this and other anatomic authors. This dense intricate plexus is concentrated in the central area of the diaphragm and thins out rapidly as it approaches the peripheral areas. It is formed by the phrenic subperitoneal branches and sympathetic branches which have their origin mainly in the cœliac plexuses. The main mass of sympathetic supply extends along the inferior phrenic artery to the under surface of the diaphragm. Numerous branches from the aortico-renal, splenic and hepatic plexuses also join in the formation. Its composite fibrils are so fine as to make it impossible to differentiate phrenic and sympathetic components, except under the microscope with differential silver nitrate staining. Numerous communications with the vagus have been described by the above authors, but their place in this study remains obscure. To this date we have failed to find reports on the proportions of phrenic and sympathetic supply in the plexus similar to that reported by Nettleship,[21] in his research on the coronary vessel innervation.

FLUOROSCOPIC DATA IN HICCUP

Routine repeated fluoroscopic examination of patients during this series has resulted in an orderly classification of events at varying intervals during the attack. Hiccup can no longer be described as a rhythmical, clonic contraction of the diaphragm as a whole. Though there is a slight divergence in a small number of cases, the majority present a definite pattern picture which may be classified as follows:

1. A single complete unilateral contraction of the whole diaphragm.
2. A single complete spasm followed by one to three smaller spasms in quick succession.
3. Contraction of one or more segments of the diaphragm, all unilateral, without complete spasm.
4. Unilateral complete spasm with segmental spasm of the opposite side.
5. Complete bilateral spasm, which may or may not be followed by smaller spasms. This type is rarely seen.

Interval fluoroscopy during the course of attacks presents a series of changes in the spasm picture. Timing of this changing sequence depends upon the severity of the attack and the condition of the patient. These changes have been observed to occur at a much more rapid rate in debilitated and exhausted patients. The importance of the differences in innervation noted above becomes self-evident in a study of this changing spasm sequence (Fig. 2).

1. A sharp pyramid like contraction of the whole diaphragmatic dome. The apex of the pyramid is always lateral to the mid line. This may continue throughout the attack.
2. A gradual flattening of the apical angle of the pyramid occurring from the fifth to the eighth day of the attack. At this stage wavy contractions of the outer segments of the diaphragm appear.
3. The pyramid is replaced by wavy contractions of the whole dome. These contractions first appear in the costal segments and proceed centrally.
4. Complete flattening of the whole dome with wavy contractions of the outer segments. This occurs late in the attack and is of grave import.

The above constitute a definite picture of progressive muscular exhaustion with gradual failure of muscular contractility.

ETIOLOGY

Based upon all the above findings, etiological classification of the causative factors in hiccup, though still presenting a complex problem, becomes much less formidable. Mayo's contribution has been discussed above. Noble Clark,[16] approaches the problem schematically, omitting the factual intermixture of many of the causative factors. His findings present a springboard for effective classification, but fail to evaluate the sequelæ in the prolonged hiccup attack. Re-

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peated mention must be made of the fact that psychogenic influence plays a great part in all but very few of the cases presenting. Even in the presence of definite causative disease, the psychogenic status of the patient must be thoroughly investigated. Our experience with these cases forced the conclusion that the severity and length of the attack depend in a direct ratio upon the psychogenic make up of the patient. Minor transient attacks are not listed.

Over a period of years, by far the greater number of cases presenting fall in the psychogenic classification. Careful enquiry and observation soon eliminate the out-and-out malingerer. Border line mental cases present a great deal of difficulty, but eventually diagnose themselves.

debilitation and emaciation of the prolonged hiccup sufferer pose a serious problem. It must be remembered that hiccup spasms may occur from forty to one hundred times per minute, and in the prolonged attack occur during sleep and even under anæsthesia. The term cerebral cortex pattern has been used in the psychiatric field as applied to these advanced cases. Though no proof can be presented in refutation of a cortical pattern, we prefer to believe that the cycle is much more likely to be centred in the hypothalamic and medullary respiratory centres. Knowledge of cortical hypothalamic pathways is at present too obscure to be of assistance in this problem, although the findings of Kuntz,[4] and Goetz,[6] pave the way for future investigation.

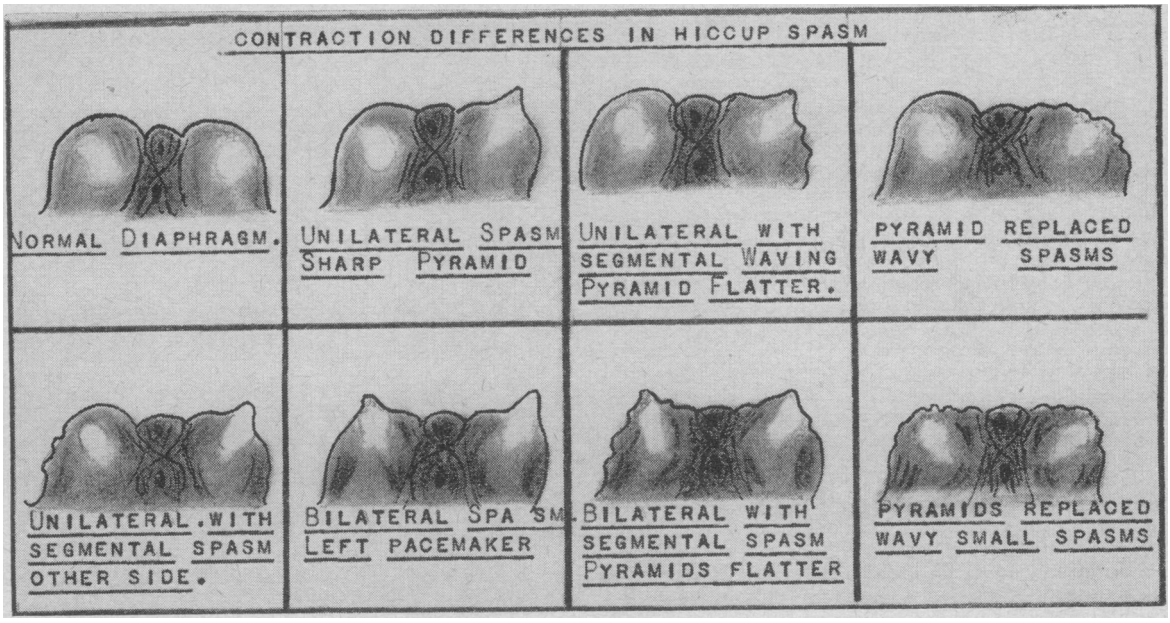

Fig. 2

The remaining patients in the psychogenic section fall into two main classes: (1) Nervous debilitated persons who have received a recent acute mental shock, or who have been under continued severe mental strain. In two of the present cases knowledge of proved sterility in one partner, discovered two years after marriage, preceded by only a short period, a prolonged attack. (2) Patients of neurotic tendency who have long complained of minor stomach or other digestive disturbances, for which no organic basis has been found. Cardiospasm and pylorospasm fall within this category.

Dope addiction has presented a small number of cases. Attacks in these cases ceased when withdrawal treatment was instituted. Extreme

TABLE I.

ETIOLOGICAL CLASSIFICATION OF PROLONGED HICCUP

Psychogenic	
Malingerers. Publicity seekers. Border line mental cases.	Mental shock. Prolonged Nervous Strain. Post partum. Cardiospasm. Pylorospasm.

To be considered in all cases with organic basis.

Organic			
Central nervous system	*Neck*	*Thorax*	*Abdomen*
Hemorrhage*	Tumours*	Aneurysm	Post-Oper/ve.
Thrombosis*	Glands	Neuroma*	Aneurysm*
Brain injury*	Aneurysm*	Tumour	Tumour
Fracture skull*	Branchial cyst	Pericarditis*	Gall bladder
Neurosyphilis*	Diverticulum	Pneumonia	Pancreatitis
Anæsthesia	Scal. Ant. Synd.	Abscess	Abscess*
Encephalitis	Arteriovenous aneurysm*	Diaph. hernia*	Ulcer
		Foreign body	Gastritis*
		Coron. thromb/s.	Foreign body*

*Cases occurring during this series.

DIAGNOSIS

The unusual classification into neck, thorax and abdomen shown in the etiological table above, is purposeful in that it covers the entire extent of the phrenic nerve, and assists materially in the elimination process leading to a diagnosis. We can all bring to mind articles published in both medical and lay press, commenting on the simplicity of phrenic crush and its use in the treatment of hiccup. Unequivocally, it must be stressed at this juncture that phrenic crush or section *per se* is not the panacea for hiccup. Experts in the tubercular and other thoracic fields have long been cognizant of the many failures of phrenic crush or simple section in controlling diaphragmatic movement. Diagnosis of any hiccup seizure of prolonged duration, must of necessity be aimed at discovery of an underlying cause, if present. Careful enquiry should elicit: (1) Duration of attack and frequency of spasm. (2) History of previous attacks and duration. (3) Psychogenic status with especial attention to recent shocks or prolonged mental strain. (4) Organic factors. (5) Treatment administered prior to present examination. The latter is of great importance due to the large number of bizarre and sometimes dangerous treatments that may have been administered. In this series of cases we have seen: third degree burns in each posterior triangle of the neck following prolonged, repeated ethyl chloride spraying (Case 3 in case histories). Extreme dyspnœa necessitating use of respirator following procaine injection of both phrenics; amytal poisoning following ill advised recommendation of administration to the comatose stage; brachycardia with cardiac collapse following prolonged strong faradic stimulation to the neck.

Determination of the type and site of the spasm is arrived at by: (1) Visual inspection of the costal arches during spasm, under a strong light. (2) Fluoroscopic examination, repeated at suitable intervals. This gives better results when the patient can be examined in the erect position. (3) General appearance of the patient with regard to emaciation, etc. In late grave cases ecchymoses are found in the lower two intercostal spaces. Ecchymosis is much greater on the affected diaphragmatic side. This sign is an indication of dire import and demands immediate drastic treatment (see treatment). (4) Phrenic circle test.

The latter test, so incorrectly described by Gazan Dzalalov,[2] in the Soviet medical press, is claimed as a first in the successful treatment of hiccup. Though it is extremely useful in ascertaining the side of the spasm, or in bilateral cases, the pacemaker side, it has no place whatever in the further treatment of the attack. This is due to the fact that spasm returns almost immediately after the test is completed. We have used the test for many years and have never noticed remission of spasm for more than a few minutes. The test is performed in the following manner:

A small pillow is placed under the patient's neck producing a slight anterior arching. The first two fingers of the right hand are placed at the posterior border of the sternomastoid muscle immediately above its clavicular insertion. The fingers are pushed medially and posteriorly at an angle of thirty degrees until the resistance of the scalenes and the transverse cervical processes can be felt. Pressure is then made directly posteriorly by bending the terminal phalanges. Pressure should be gentle and steady and should not exceed two minutes. Spasm usually ceases on the pressure side within one minute of the application. Testing one side after the other simplifies the determination of uni- or bilateral spasm. Where bilateral spasm is found, in all cases seen, spasm is observed to be greater on one side than the other. This has been termed the pace-maker side. Treatment should be directed only to this side. We have found that successful treatment of the pacemaker side, (where surgical treatment has been decided upon), is followed by subsidence of spasm of the opposite side. Bilateral cases are however, rare.

TABLE II.

CASE STATISTICS

Unilat.	*Bilat.*	*Etiology*	*Duration*	*Male*	*Female*	*Findings*
15	4	No pathology	7-60 dys.	2	12	Psychogenic*
4	1	Slight "	6-27 dys.	2	3	"
4	2	Postoper/ve	3-8 dys.	5	1	Abscess 2 Obstruction 2 Perforation 2
1	0	Organic	36 dys.	1	0	Neurofibroma phrenic nerve*
4	0	Organic Brain. Skull	5-23 dys.	4	0	Hæmorrhage* Tumour. Injury* Fracture*
1	0	Organic Aorta	26 dys.	1	0	Aneurysm following wiring*
0	1	Organic Stomach	13 dys.	0	1	Swallowed foreign body
1	0	Organic	15 dys.	1	0	Neurosyphilis controlled*
0	2	Organic	5-9 dys.	0	2	Scalenus Ant. Syndrome
0	2	Post partum	6-22 dys.	0	2	Psychogenic*

*Cases described in case histories below. Total cases 42.

TREATMENT

Any mode of treatment must be based upon a careful evaluation of all factors concerned. Study of the statistical chart above emphasizes the fact that most cases are of long duration. It is important to remember that a history of all

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treatments administered should be obtained before instituting any further therapeusis. We cannot stress too strongly that early rushing into surgical intervention is far from the intent or purpose of this present article. Every therapeutic aid recommended should be tried, and in some cases repeated, and one must be constantly alert for any etiological factor that may have been missed in earlier examinations. Only when all advances have failed, and the rapid deterioration of the patient becomes of grave concern, is surgical intervention advised. Debilitation and emaciation should, however, not be allowed to proceed to the stage where any type of surgical shock may have dire results. The advent of antibiotics has drastically reduced the number of postoperative cases seen. Where postoperative hiccup does occur, the pathological basis must be sought for and eliminated. We do not agree with Noble Clark,[17] that hiccups occurring after the third postoperative day are of grave import. A brief discussion of published treatments is of value in determining the therapeutic *modus operandi* of the first days of the attack. Should these and other measures fail, treatments indicated below should be instituted without delay, in an effort to forestall the rapid deterioration of the patient which sets in after this period.

Benzedrine sulphate.—Shaine Marks[17] quotes results in a small number of cases, all of which were postoperative and of short duration. Trial of this drug in other types of attacks has proved ineffective. Not recommended.

Quinidine sulphate.—Bellet and Nadler[18] report a number of cardiovascular and uræmic patients, a percentage of whom appear to have been *in extremis*. The dosage of the drug appears frequent and large. Information from the cardiological point of view is that in many cases this may be dangerous dosage. Ineffective in prolonged cases.

Carbon dioxide and oxygen. — Hamilton Bailey.[19] Administration of these gases has proved of definite value in the early treatment of hiccup. It has not proved helpful in later cases and in our opinion should only be used in the first three days. It is strongly advised (following some unfortunate sequelæ in injudicious use) that this be administered by a competent anæsthetist. Periods of inhalation should not exceed ten minutes. It is not recommended in extremely debilitated or emaciated patients. Open phrenic nerve injection as recommended by this author, has proved of doubtful and only temporary value. Bilateral injection should never be performed.

Barbiturates.—Many authors strongly recommend the use of this drug. Though it has proved extremely useful in early cases, excessive dosage and its use in late hiccup is to be discouraged. The recommendation of one author to push this drug to the comatose stage needs no further comment.

Galvanic sinusoidal current.—Barnard.[20] The application of galvanic current as outlined by this author, has, in his hands, met with success in a limited number of cases. Those who, in the wave of enthusiasm that followed his publication, have tried this procedure have been disappointed in its results. There is no doubt, however, that repeated applications in the early stage will in a considerable number of cases slow down the rate of hiccup. In the later stages of the attack the application becomes intolerable to an already debilitated patient, and has shown little if any effect.

Ethyl chloride spray.—Though still recommended by some, this treatment is completely useless. Sad results of prolonged spraying of both sides of the neck are described in Case 3 in our case histories below.

Psychiatric treatment. — Hypnosis has been recommended and tried. There are no reported sucesses. Except where there is a question of actual mental disease, consultation can give little assistance in the acute phase.

Deep anæsthesia.—Tried on numerous occasions. Completely useless and not recommended.

Space does not permit mention of the hundreds of varied treatments suggested and tried without any success.

Cases seen early in the attack should be placed on the following regimen: (1) Complete restriction of oral feeding or drinking. (2) Intravenous saline and glucose up to a maximum of 3,500 c.c. in 24 hours. Protein and vitamin therapy where indicated. (3) Thorough bowel cleaning by S.S.E. (4) Absolute visitor restriction. (5) Complete laboratory check. (6) Sodium amytal gr. 3, q.4 h. for 24 hours only. (7) Fluoroscopy once during the first day.

The above should be carried out for 24 hours. If hiccup is relieved or slowed down, continue with intravenous fluids for another 24 hours without further medication. With no relief, continue intravenous fluids for a third 24 hours,

with demerol mgm. 100, q.6 h. Fluoroscopy is repeated during this period. Galvanic current is applied as per method of Barnard at intervals during this 24 hours. If the first three applications of the current show no results, application is discontinued. Carbon dioxide and oxygen, at intervals of at least three hours during the ensuing 24 hours, should be administered by a competent anæsthetist. Periods of inhalation must not exceed ten minutes.

With failure of all the above intravenous therapy is discontinued, and the patient placed upon a semi-soft diet. One or more of the above treatments are repeated. Surgical intervention should not be delayed after the seventh day. Experience has shown rapid deterioration of the patient after this period. Fluoroscopy, to evaluate any changes in the spasm picture, is of great assistance in formulating the extent of intervention that may be required. General supportive treatment is maintained throughout.

SURGICAL INTERVENTION

In considering the anatomical data given above, it becomes evident that any surgical intervention must encompass all sections of the anatomic picture. Therefore the plan of action must include the following succession of moves, any one of which may be successful in abating the attack, but in which it may become necessary for all to be carried out before cessation of the attack will occur. (1) phrenic circle interruption. (2) Sympathetic interruption. (3) Lower thoracic and upper lumbar nerve interruption.

Following an appreciable number of failures of phrenic crush or simple section to control spasm, we have devised a technique of complete phrenic dissection which has proved successful in 95% of cases. In a small number of cases, although the major spasms were relieved, segmental spasm of the peripheral diaphragmatic areas continued. Control of these segmental spasms was attained through 2 and 3 above.

PHRENIC DIVISION

The nerve is approached by an incision extending from the posterior border of the sternomastoid, two fingersbreadth above the sternoclavicular joint, for about two inches posteriorly. The main trunk is easily recognized on the anterior surface of the scalene muscle. A portion of the nerve is dissected free from the prevertebral fascia. A hook is passed beneath this portion and the nerve is gently lifted away from the muscle surface. Careful dissection is carried upwards until the cervical roots are exposed. Continuing gentle traction on the nerve trunk, dissection is then carried down to the clavicle level. During this dissection any abnormality in root formation becomes quickly discernible. Mention is repeated of an abnormal cervical 5 root, medial to the main trunk, which may extend to the superior mediastinum, and must be differentiated from the sympathetic chain. All cervical roots are now crushed. The majority of the small fibril-like communicating branches to the vagus and sympathetic chain will be found at the level of the superior and inferior cervical ganglions. These are cut or simply pulled away. Finally the main trunk is crushed at the lowest level seen. The wound is then closed without drainage.

The above procedure usually results in complete cessation of the hiccup spasm. In three of the more severe cases, although there were no noticeable or auditory signs, patients complained that they still felt they were hiccuping. Fluoroscopic examination revealed small segmental peripheral spasms. Procaine injection paravertebral, of thoracic ganglia 8 to 12, with injection of eleventh and twelfth thoracic effectively stopped these smaller spasms. Paravertebral sympathetic injection without phrenic dissection was tried in some of our cases. Though the frequency and severity of the spasm was reduced, we were not successful in obtaining complete cessation. Increasing the area of injection from thoracic 6 to thoracic 12 showed no improvement over the shorter injection. There have been no deaths in this series. Recurrences took place in five cases, one to three years after operation. The attacks, however, were mild and responded to ordinary treatment.

CASE HISTORIES

Cases here presented were selected because of wide differences in their etiological factor.

CASE 1

Miss A.M., white female, age 19. Previous history negative. Length of attack 23 days. Spasms per minute 60. Weight 83 lb. Normal weight 125 lb. Condition on admission: pulse 120, respiration 30, temp. 99.4°. Examination negative except for slight fullness in left posterior triangle. Spasm, left unilateral. Patient states she had had slight hiccup attacks, lasting from an hour to a day during the past six months. Present attack started after a cold and has increased in intensity. Operation revealed a neurofibroma in the central cervical portion of the phrenic nerve. Removal resulted in complete cessation of the attack. Two years later, 1942, another severe attack developed lasting 52 days. Patient complained at this time of long continued pain in the left chest. Spasm, unilateral and left-sided. X-ray showed negative except for an olive shaped bulging of the left pericardium. In view of the previous history, thorax was opened, and a neurofibroma of the phrenic nerve, about the size of an olive, was found in the mid pericardial section of the phrenic nerve. This was removed with complete relief.

CASE 2

Mr. H.K. Ordained Rabbi. Age 34. Previous history negative. Married two years. Advised by his doctor to have semen examination because of failure of his wife to become pregnant. Two weeks after receiving a sterile

semen report, he began to hiccup. In spite of the spasm he continued with his duties for five days. Following this he was treated for fourteen days with most of the remedies outlined above. Temperature 100°, pulse 140, respiration too rapid and jerky to be counted. Spasm 50 per minute. Bilateral, with stronger contractions of the left diaphragm. Left phrenic dissection April 1948. Immediate cessation. No recurrence. Regular duties since discharge.

CASE 3

Mrs. B.F., age 25, white. Previous history negative except for constant fear of delivering an abnormal child. Two members of her immediate family had delivered hydrocephalic children. Delivery of a normal child two months previous to onset of hiccup attack. Patient admitted to another hospital seven days after attack commenced. Narcosis, deep anæsthesia and repeated sprays of ethyl chloride to each side of the neck. When seen 14 days after inception of attack, temp. 101°, pulse 140. Respirations too rapid and jerky to be counted. Weight 132 lb. Normal weight 158 lb. On each side of the neck there is a large deep grayish looking ulcer involving most of the posterior triangle. Hiccup spasm 60 per minute, bilateral with stronger contractions of the left diaphragm. Condition of patient very poor. Left phrenic dissection September 1948. Immediate cessation. No recurrence.

CASE 4

R.G., age 67, white male. Two months prior to admission had undergone wiring of an aneurysm of the abdominal aorta by acknowledged expert in this field. Hiccup spasm commenced three days after operation. Spasms were slight during next few days. Patient was readmitted to hospital one week after discharge because of increase in severity of attack. All recommended treatments were tried. On admission to our service—Temp. 98. Pulse 64. Respiration 22. Spasm rate, 45 per minute. Left unilateral. X-ray disclosed loose coil of wire in the region of the cœliac plexus. Because of poor condition and previous surgery, it was deemed inadvisable to attempt to remove wire. Sympathetic paravertebral block of thoracic 7 to 12 reduced rate of spasm to ten per minute. Phrenic dissection December 1951. Complete relief. Died 18 months later.

CASE 5

J.T., age 48, while male. Treated for syphilis twenty years ago. Treated for neurosyphilis up to ten years before attack. No symptoms for the past five years. Head of successful large business. Present attack started two weeks before examination. Admitted to hospital on neurological service. On the twelfth day he developed severe hæmatemesis. We have twice seen this complication and believe that this is due to rupture of the cardio-œsophageal veins, following œdema and engorgement due to the constant tension and pull of the diaphragm in spasm. On examination, temp. 99°, pulse 130, respiration 30. Patient critically ill. Rate of spasm 45 per minute. Visual fibrillary spasm in lower two left intercostal spaces. Spasm, left unilateral with small wavy spasm of right diaphragm. Wassermann negative. Argyll Robertson pupils otherwise no evident signs of neurosyphilis. Phrenic dissection followed by paravertebral sympathetic injection and injection of thoracic 11 and 12. Hæmatemesis ceased immediately after phrenic dissection. Discharged December 1951. No recurrence to date.

CASE 6

E.K., age 60. Practising physician in New York. 14 days previously patient was admitted for cerebral accident. Coma 48 hours. Recovery after third day good. Vertigo and changing areas of anæsthesia and paralysis led to diagnosis of mid brain hæmorrhage by medical service. Hiccup spasm commenced on eighth day following cerebral episode. During the ensuing 48 hours rate of spasm increased and patient began to go downhill rapidly. No treatment was given because of cerebral history. On examination; patient in poor condition. Weakness but no absolute paralysis of both legs and left arm. Anæsthetic areas in right leg and left arm. Spasm rate 45 per minute. Spasm, bilateral with stronger left contraction. Phrenic dissection slowed down spasm to ten per minute. Paravertebral block and thoracic nerve injection caused almost complete cessation except for small spasm in the outer segments of the left diaphragm. Intercostal injection repeated. Spasms controlled. Patient discharged December 1950. No recurrence. Has now resumed practice.

CASE 7

C.K., age 38, white male. Basal skull fracture, fracture maxilla and mandible sustained in automobile accident. Condition grave on admission but after stormy period making good recovery. On fourteenth day patient fully conscious, no paralysis. Marked diplopiā. On eighteenth day hiccup attack began and increased in severity at a rapid pace. Because of skull fracture and possible brain injury, phrenic dissection under local anæsthesia. Complete cessation of all spasm. Discharged March 1949. No recurrence. Diplopia persisted for about eighteen months after discharge. No recurrence of hiccup. Phrenic dissection under local anæsthesia proved very difficult and is not recommended. Tugging on the nerve is badly tolerated.

SUMMARY

This article reviews the subject matter based upon a ten year study, embracing:

1. Fundamental advances in anatomical knowledge of muscle formation and innervation of the diaphragm.
2. Present day concept of phrenic nerve anatomy, and similar advances in the structure of sympathetic areas of distribution.
3. Mechanics of the hiccup spasm.
4. Etiological classification.
5. Statistics and case histories.
6. Suggested treatment and results.

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