Much has been said and written which appears to be vastly concerned with the matter of unjust compensation awards as applying to the interests of the insurance companies. The safeguarding of the claimant, apparently a negative consideration, is perhaps decidedly quixotic. Nevertheless, it must in all fairness be conceded that insurance companies undertake the risk of compensation for monetary consideration. It would appear in the face of the foregoing that cold, unemotional logic points to awarding for the claimant in the face of a definite and reasonable degree of suspicion, inasmuch as proof is often impossible of attainment. Unfortunately it sometimes appears that the reverse obtains and it is perhaps to be expected under circumstances which obviously demand a greater degree of courage to affirm claims than to negative them.

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STUART C. WAY, M. D. (490 Post Street, San Francisco).—This paper brings to our attention a subject, endlessly disputed, whose solution lies in the distant future, if at all.

Certainly much could be done in simplifying the problem by making:

1. *A Correct and Early Diagnosis.*—Trained specialists alone can accomplish this, by a careful review of each case, giving the necessary attention not only to the irritants encountered at work, but those at home. The age of the patient, the type of skin, internal factors such as food and an accurate history of previous skin trouble all enter into the solution of the problem.

2. *Thorough Laboratory Examinations.*—The Kline precipitation test now renders the exclusion of lues a matter of minutes and is proving very reliable. Gummata due to traumatism are often unrecognized. Scabies, too, is responsible for many of our cases of alleged cement, lettuce and poison oak dermatitis.

3. *The Patch test* has its limitations, but some of these can be overcome by recalling that susceptibility is not a permanent state but subject to internal and external influences such as diet, focal infection and the elements. Also sensitization may be present in one area of the skin and absent in other areas.

4. *Compensation* in the ergodermatoses should be governed by the actual damage done to the patient rather than by the duration of the injury which is often prolonged by extraneous factors, overtreatment, etc. Because of increased vulnerability of the skin after forty, age becomes an important factor and accounts for the chronicity of many dermatoses. Recurrences are sometimes accounted for by home contacts.

5. *Disputes* in handling and in the status of industrial dermatoses could be appreciably reduced by a careful rating of the worker according to age, physical condition and previous skin troubles.

6. *Protection* against irritants encountered in the industrial world should be studied by qualified men and the knowledge gained imparted to those concerned.

Insurance companies should realize that it is much less expensive in the end to have dermatologic cases seen by qualified, experienced dermatologists. Disability is often prolonged by faulty diagnosis and maltreatment.

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STANLEY O. CHAMBERS, M. D. (1260 Roosevelt Building, Los Angeles).—The absence of an etiologic understanding in many common dermatoses leads to theoretical, though just conclusions. It is often necessary in dermatologic practice to associate a clinical picture with environment in determining causation. This is particularly true in the study of so-called industrial or occupational skin diseases. Our medical fathers had no alternative. Methods of investigation had not been identified. Medical progress, however, has opened a field which promises even greater practicality and accuracy than has so far been developed. This new phase of investigation in occupational dermatoses has been well brought out in Doctor Scholtz's discussion. The clinician now need not only suspect a relation between cause and effect, but with little difficulty can demonstrate it.

The clinical similarity between certain mycotic infections and so-called diathetic eczema to that dermatitis produced by contact irritants has undoubtedly, in the past, lead to error and unjust compensation.

EPILEPSY IN CHILDREN*

WITH PARTICULAR REFERENCE TO THE KETOGENIC DIET

By HOWARD R. COODER, M. D.
Los Angeles

DISCUSSION *by J. M. Nielsen, M. D., Los Angeles; William Palmer Lucas, M. D., San Francisco; Henry Douglas Eaton, M. D., Los Angeles.*

THE following report is based upon one hundred cases of epilepsy in children up to twelve years of age treated in a special clinic of the Los Angeles Children's Hospital during the past three years. The proportion of these one hundred cases to a total of seventeen thousand admissions to the General Clinic is 0.6 per cent, which corresponds to statements that the incidence of epilepsy in children is from one-half to one per cent. Our one hundred cases have been classified as follows:

Major epilepsy (grand mal)	52
Major and minor epilepsy	16
Minor epilepsy (petit mal)	14
Jacksonian epilepsy	6
Pyknolepsy	4
Hystero-epilepsy	2
Epileptic equivalents	6

Major Epilepsy.—Major epilepsy includes the majority of the cases. The onset is sudden, sometimes with a warning, or aura, in a very few with a cry. In nearly all major cases loss of consciousness is early and complete. The child may fall, but as many children have their attacks only at night or during naps or while playing on the floor, the fall is not always a part of the attack. Tonus and clonus may supervene in order, but more frequently occur together in quite irregular fashion. Attacks last from one to five minutes in most cases, occasionally longer. Sleep of an hour or less usually follows and a few children are fretful or otherwise perturbed for a few hours to a day afterward, but many recover quickly and go about their activities without showing that they have had a convulsion.

A number of our patients have both major and minor attacks, sometimes frequent minor with an occasional severe attack, sometimes an equal number of both. A few who formerly had only one type now have only the other type; it happens that of our patients there are more who have passed from a former major epilepsy to a present minor epilepsy.

Petit Mal.—The minor type of epilepsy, or petit mal, includes a large number of transient seizures,

* Read before the Pediatric Section of the California Medical Association at the sixty-first annual session, Pasadena, May 2–5, 1932.

spells, lapses of short duration, from a few seconds to a minute. There may or may not be loss of consciousness, the eyes roll up, a passing shudder or stiffness occurs. Although the minor attack is not nearly so long or so severe as a major seizure, the postconvulsive sequelae of sleep, mental confusion, or fretfulness are just as likely to occur, and it appears that the ultimate effect upon the personality and mentality of the child are as much to be feared from the one variety as from the other.

Jacksonian Epilepsy. — Jacksonian epilepsy in our six cases has conformed to the classical description of spasm beginning in an extremity of one side and successively involving the muscle groups of that side, consciousness not being lost until the convulsion has become generalized.

Pyknolepsy.—Pyknolepsy is remarkable for the great number of spasms of a very minor nature which may occur in one day, as many as a hundred or more. They consist of a transitory staring, turning up of the eyes, blankness, stopping of activity, of not more than ten seconds' duration. It is difficult to say if there is loss of consciousness; there is never any convulsion. The condition is described by Sachs [1] and also by Thomson [2] in almost identical words. It is very resistant to treatment, but tends to disappear by itself after more or less time. It is probably not epilepsy at all, nevertheless a case of it deserves to be watched until it has been differentiated from the more serious disorder.

Hystero-Epilepsy.—Hystero-epilepsy describes seizures which are identical with epilepsy in many ways, but which occur only under circumstances of stress, emotion, or while the patient is under examination, a thing which seldom is true of genuine epilepsy. It is not nearly so common in children under ten as in those of adolescent years or older. We have had two cases.

Epileptic Equivalents. — Epileptic equivalents include a number of conditions other than seizures which, by their periodic occurrence, their involuntary nature and occasional development later into true epilepsy, require treatment as for the latter. Attacks of mental confusion, transient changes in disposition, certain hallucinations similar to aurae, and migraine, may be considered under this heading.

ETIOLOGY

For many years neurologists and pathologists have sought unsuccessfully for a characteristic lesion of brain or nerve substance. More recently the causes of epilepsy have been pursued in the avenues of biochemistry, endocrinology, psychology, without an answer to the problem. Etiology is thoroughly reviewed in the monograph by Lennox and Cobb.[3] There are many theories of cause, no one much better than another. As these authors say, we are in need, not of more theories, but of more facts. Researches like the work of L. O. Morgan,[4] who has produced epilepsy in animals by injecting destructive substances into certain nuclei at the base of the brain, are a definite help. In the end it may come, as Hughlings-Jackson said forty years ago, to the fine point of recognizing the difference between cells which react with convulsions and cells which do not.

TREATMENT

The really great advances in the treatment of epilepsy have been made in the last ten years by the researches of biochemistry and metabolism. They include: fasting, dehydration, and the ketogenic diet.

These methods have been suggested by clinical observation and laboratory study of body conditions before and after fits, by attempts to produce sedation, and by careful correlation of the successful results of all forms of treatment.

Fasting.—Fasting is one of the many forms of treatment which has been in more or less use for years, but it received its first serious examination by Geyelin [5] in 1921, who studied twenty-six cases upon fasts of twenty days. Fasting is an effective dehydrating measure and causes ketosis. As a sole method of therapy it is discountenanced by some workers. In small children it certainly cannot be maintained for more than a few days at a time. In cases which we have selected for special study we have found that a five-day fast is an excellent way to introduce the ketogenic diet.

Dehydration.—For a long time the finding of edema of the brains of epileptics at autopsy, and of "wet brains" when epileptic patients were operated upon for decompression or exploration, has focused attention upon the possible advantages of limiting the water intake of these subjects. More recently this matter has been studied by McQuarrie,[6] who has been able to effect cessation of convulsions by restricting water and increase of convulsions by forcing fluids. It is a form of treatment difficult to maintain with children, unless they are in bed, because the fluids have to be limited to no more than 200 to 300 cubic centimeters per day. As a single method of treatment, we do not consider it superior to the ketogenic diet, and we have shown in some twelve to fifteen cases kept in the hospital for a trial period that the diet will produce just as effective dehydration as water restriction. Dehydration is, nevertheless, an important underlying cause or result in several forms of treatment and deserves consideration.

The Ketogenic Diet.—This was developed in 1921 by Wilder [7] from his experiments with fasting, in order that he might prolong ketosis which he had noticed was a constant occurrence in patients upon a fast. The ketone bodies, acetone, aceto-acetic acid, and b-oxybutyric acid are produced in the body under certain abnormal conditions, as the acute febrile illnesses of children; also they occur promptly upon a complete fast and upon a diet high in fat and low in carbohydrate. In the latter case it is the incomplete hydrolysis of excess fat which results in the ketone bodies. Persons vary in their ability to burn fat, and of a number of children upon the same ratio of ketogenic diet the majority will show a strong acetone reaction in the urine, while a small number will exhibit a slight or inconstant excretion of acetone. Heinbecker,[8] working with a group of Eskimos

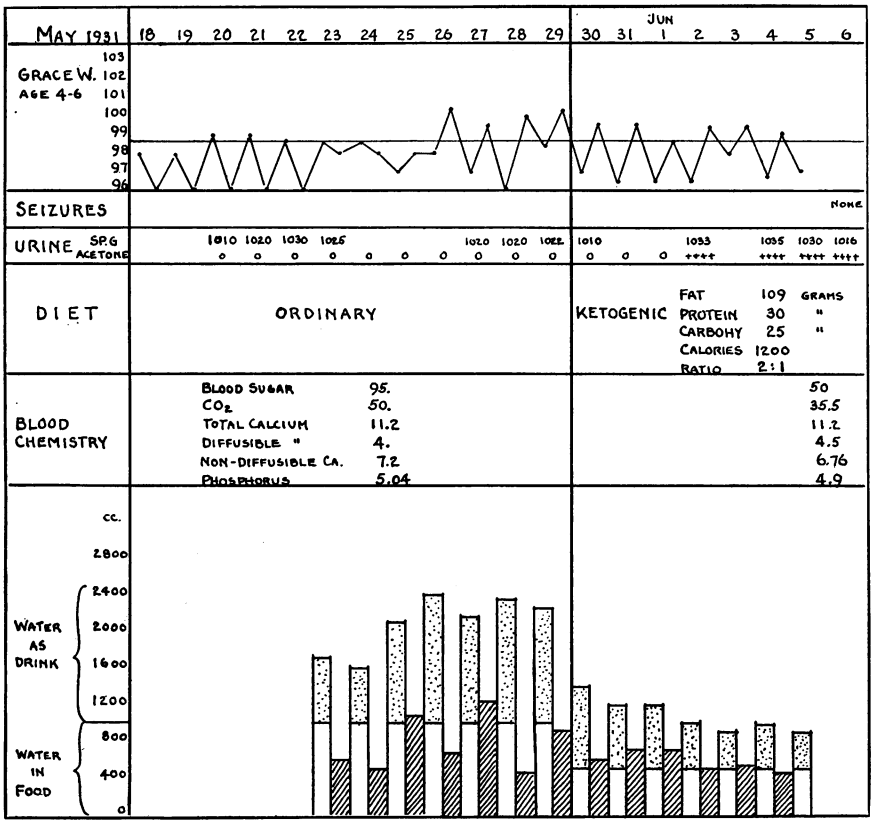

Chart 1.—Chart showing the prompt reduction of water intake and output upon beginning the Ketogenic Diet.

upon experimental starvation, found that they developed no ketosis whatever.

Ketosis is present for months at a time in our patients without presenting any of the unfavorable symptoms of its related state, acidosis. Of ten cases upon whom the CO_2 combining power of the plasma has been determined, only one showed a low figure (35 vols. per cent); there was, however, no clinical evidence of acidosis.

Prescribing a ketogenic diet is greatly facilitated by the use of a table such as that in the book by Talbot,[9] which gives a complete discussion and instructions. We set the figure for protein at a minimum of 1.5 grams per kilogram of body weight. The majority of children do not complain, and only two of one hundred have been made ill by the unusual food. Much of the success of this work is due to the skill of our special dietitian in designing various alternatives and frequent changes, so that the children are kept interested.

Within three days acetone appears in the urine and its presence is used as a test of whether the patient is eating all of his diet and is eating nothing else. It is an easy test and is fairly reliable; nevertheless a few of our patients who have been very careful, and who have been free of attacks for a long time, have seldom or never showed any acetone in the urine. Within the first week there is nearly always a loss of three to five pounds weight, due to dehydration. The patients all soon show physical improvement, better color, better expression and tissue turgor, and a number of minor betterments are usually reported by the parents, many of which are merely the results of a more systematic way of living. The patients report to the clinic at first once a week, then every two weeks, and when attacks are finally stopped, once a month. After a year of freedom from seizures we allow them to resume gradually an ordinary diet. Of our series there have been no relapses, but our number is too small to make a pronouncement upon this matter.

The ketogenic diet has now been in use for nearly ten years,[10] and it may fairlly be said to be the most valuable single therapeutic measure in the present list for the treatment of epilepsy. Its superiority is not only because of its greater effectiveness, but because it can be continuously maintained for an indefinite period, while some of the other forms of treatment cannot be.

Phenobarbital (Luminal).—We use this drug only as a supplement to the ketogenic diet when the latter has been on trial for three months without producing improvement in attacks. Helmholz and Keith[10] have stated that these two forms of treatment often effect a better result than either one alone. We give one, or one and one-half grain doses at bedtime, and occasionally an additional three-fourths grain in the morning. There have been no disturbing symptoms with phenobarbital as high as three grains per day over periods of many months. Only one child developed a rash.

Clinical Management.—It is important to use persistence in treating epilepsy, either as private patients or in clinics. Our successful patients may roughly be divided into three groups: those whose attacks stop promptly and completely at the commencement of the diet; those whose attacks gradually diminish to zero; and those upon whom the treatment seems to make no effect until weeks or months later, when their seizures may be suddenly and effectively terminated. It does not seem to be possible to foretell what will be the success of any one case.

If a patient misses two visits to the clinic, a social service worker is sent to use every effort to get him to return. Of fifty-five patients who have left our clinic before being discharged, we have reports on the present condition of forty-seven; only one is said to be free of attacks, the majority are still having them, and a few are im-

proved. This shows that with those who have left treatment there has not been a trend to spontaneous recovery.

ANALYSIS OF RESULTS

Total patients received in three years by the clinic, 100.

Patients at present under observation, 45.

Recent patients (less than three months), 7.

Patients treated more than three months, 38.

Patients free of seizures more than three months, 19, or 50 per cent.

Patients improved, 13, or 34 per cent.

Patients not improved, 6, or 16 per cent.

SUMMARY

A report is presented of one hundred cases of epilepsy treated in a special clinic of the Los Angeles Children's Hospital.

A distribution is made of these cases into clinical varieties of epileptic seizures, which are described.

Etiology is still in the theoretical state and is not discussed in this paper.

Of treatment the metabolic methods are described. Fasting is effective but impractical. Dehydration is important and may be the underlying factor in all successful treatment. Water restriction is difficult to carry out. The ketogenic diet has been the method of choice because of its superior effect and its practical nature. Phenobarbital has been used in conjunction with the diet.

Some points of clinical management are emphasized.

An analysis of results is presented.

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DISCUSSION

J. M. Nielsen, M. D. (1253 Roosevelt Building, Los Angeles).—It has been my privilege to see many of these patients with the author for the purpose of separating those with organic central nervous system pathology from the so-called idiopathic group. In a considerable percentage of patients one can discover evidence of brain damage: birth trauma, encephalitis, meningovascular lues, various degrees of agenesis, etc. When the condition of the patient justifies it surgical intervention is attempted; but when years have elapsed since an obvious injury surgery is ordinarily useless and the treatment resolves itself into the treatment of idiopathic epilepsy (symptomatic therapy). It is along this line that the author has done a great deal of work.

The surprising fact has been demonstrated that a symptomatic epilepsy responds to dehydration and ketogenic diet just as well as or even better than an idiopathic one.

In adults dehydration is apparently the most potent single factor in the treatment. But in childhood, in cases of home treatment, it is almost impossible to carry out. We have gone to the extreme of removing all faucets in the home, only to have the child steal water outside. In our private practice we urge restriction of fluid intake, but do not rigorously enforce it in children. On the other hand, the ketogenic diet which does relatively little good in adults is a potent remedy in childhood, as the paper well demonstrates.

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William Palmer Lucas, M. D. (490 Post Street, San Francisco).—Doctor Cooder's analysis of the one hundred cases of epilepsy which they have treated at the special clinic of the Los Angeles Children's Hospital represents a fine piece of clinical research investigation. It is from this type of work that we get our most accurate and fundamental knowledge of these most difficult cases. It is entirely a different problem to straighten out a case of epilepsy in the hospital, where you have the patients under absolute control, than when they are at home, or supervised from a clinic.

In regard to the treatment advocated by Doctor Cooder, I think he has been very conservative in his estimate of the various types of treatment. There is no question but that the ketogenic diet in many cases has proved its value. It is very much more difficult to carry out restriction of water intake in private practice and in clinic patients than it is while the patient is in a hospital bed, where absolute control can be kept over the water intake. However, with coöperative patients, a good many will stay on the low fluid intake. These patients usually are the ones that show the greatest reduction in the number of seizures, but unless we have the coöperation of the family and the child it is almost impossible to carry out the dehydration treatment.

I think everybody is of the opinion that phenobarbital should be used in conjunction with both the dietary and dehydration treatment.

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Henry Douglas Eaton, M. D. (1136 West Sixth Street, Los Angeles).—The treatment of epilepsy has always been a discouraging matter. Ketosis and dehydration have unquestionably demonstrated their value as therapeutic agents. Doctor Cooder's excellent paper proves the practicability of using this type of treatment successfully in an out-patient clinic. His results compare very favorably with those obtained with hospital control of the patient and with results in private cases, where one would perhaps expect more intelligent coöperation from the families.

In my own practice I do not use the ketogenic diet in adults, but find it valuable in children. In adults, fluid restriction, combined with medication, has proven of most value. Medication alone will control convulsions in a definite percentage of cases without apparent deleterious effects.

The author is to be congratulated on a careful and valuable clinical study. It is to be hoped that he will publish his further observations.

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Doctor Cooder, (Closing).—All of the doctors, in discussing this paper, have stressed dehydration as a form of treatment. It is not agreed by all investigators, however, that dehydration is the final factor which brings about the cure of epilepsy. Following water loss there may be more particular changes, *e. g.*,

alterations in mineral balance, as has been brought out by the work of Bridge and Iob. Voluntary limitation of water intake is difficult to teach to children, and its enforcement in a large clinic throws too much dependence upon the self-control of the little patients. It has been shown that the ketogenic diet brings about a lessened intake of water equal to what can be accomplished with fluid limitation and with no persuasive effort.

The ketogenic diet has its percentage of failures, but it is the most successful single therapeutic measure today and it can be safely continued over a long time.

I agree with Doctor Nielsen that cases of Jacksonian epilepsy, in spite of their one-sided and focal manifestations, respond just as well to this treatment as the generalized, idiopathic type.

ACUTE PERFORATED PEPTIC ULCERS*

A CLINICAL REVIEW OF ONE HUNDRED AND FIFTY-FIVE CONSECUTIVE PATIENTS TREATED SURGICALLY

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AND
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DISCUSSION *by Burns Chaffee, M. D., Long Beach; John Homer Woolsey, M.D., San Francisco; Thomas O. Burger, M. D., San Diego.*

ACCORDING to Watson,[1] the earliest account of a perforated gastric ulcer was presented to the Royal Society in 1729 by Christopher Rawlinson. In this country O'Hara [2] reported the first example to the Philadelphia Pathological Society in 1875. The first successful surgical excision of a perforated duodenal ulcer was reported by Dean [3] in 1894. Since that date an ever-increasing amount of literature has accumulated which crystallizes our thoughts as to early diagnosis and treatment.

The subject-matter for our present study was obtained from a careful clinical review of 155 consecutive patients with acute perforated peptic ulcer. These patients were treated surgically by the San Francisco Emergency Hospital Service.

What is the probable incidence of acute perforations among patients with a proven peptic ulcer? In 1911 W. J. Mayo [4] reported the occurrence of twenty-five perforations among a series of 272 proven peptic ulcers (10.9 per cent). Trout [5] in 1928 reported a definite increase in acute perforated ulcers among the enlisted men of the United States Army stationed at Hawaii. The incidence increased from one in forty-one (2.5 per cent) in 1922 to one in seven in 1926 (14.4 per cent). Such statements show that the danger of perforation is a factor of considerable importance.

CENSUS OF THE GROUP

This disease is found most frequently in the lower social scale of life, for these individuals as a class are less likely to have proper dietary and medical supervision for their indigestion. This type of patient also is more likely to have poor oral hygiene, and other foci of infection.

Perforated peptic ulcer may occur at the extremes of life. Cecil Finney reports a case in an infant two months old. Our oldest patient was a man of seventy-two years of age. Our series shows the disease to be one of early adult life. The average age was forty years, while about 60 per cent of the patients were in the third and fourth decades of life. Ninety-five per cent of the patients in our series were males.

FACTORS PROBABLY CONTRIBUTING TO THE CAUSE OF ACUTE PERFORATION

1. *Family or Individual Predisposition to Development of Ulcer.*—Among our patients there were three instances where other members of the family had acute perforated ulcers. Two patients reported in this series have each had three emergency operations for this same disease.

2. *Septic Foci and Upper Respiratory Infection.*—It was very noticeable that the majority of these patients had poor oral hygiene, which many investigators believe to be an initiating or aggravating factor in the disease. Of equal significance was the apparent seasonal variation, for 70 per cent of our patients were admitted during the six-month period of winter and spring when acute upper respiratory infections are most prevalent.

3. *Improper Dietary and Medical Supervision.* The social status of these patients as a group precludes satisfactory dietary regimen and intelligent medical supervision of their ulcer problem.

4. *Indiscretion of Diet and Alcoholism.*—In many patients of this series, perforation occurred immediately following an eating or alcoholic drinking orgy. One patient suffered perforation after complying with a fake ulcer "cure" which consisted of a twenty-one-day fast broken by the ingestion of one gallon of milk.

5. *Sustained Physical Effort.*—There seems to be some relation between sustained physical effort and exacerbation of ulcer symptoms and perforation. In several patients of this group, perforation occurred suddenly during the hard physical exertion required by their occupation, but they were unsuccessful in contesting their cases before the Industrial Accident Commission.

CLINICAL PICTURE

Each of us no doubt can recall vividly our first patient suffering from acute perforated peptic ulcer. The initial symptoms and signs may be reviewed by quoting Moynihan's accurate and vividly expressed observation of the catastrophe:

"When perforation occurs there is a sudden onset of the most intolerable agonizing pain. The pain is hardly exceeded in severity by any that a human being can suffer; the extremity of agony is reached. So profound may the instant impression be that death results. . . . The patient is always prostrate with agony; the eyes are wide and watchful; beads of sweat stand out upon the brow and the lines are quickly graven on the cheeks. The patient breathes shortly and quickly; he cannot take a deep inspiration. . . . the attempt to do so ends in a groan or shout of agony and a spasm of pain. The answers to one's questions are given in snatches and every expiratory phase ends

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* From the department of surgery, University of California Medical School, and the Department of Health, San Francisco, California.

* Read before the General Surgery Section of the California Medical Association at the sixty-first annual session, Pasadena, May 2–5, 1932.