DIAGNOSIS AND TREATMENT OF HYPOTENSION DURING ANESTHESIA * †

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The subject of cardiac arrest in all its ramifications has received vigorous treatment in recent years (1–3), and rightly so. However, other aspects of abnormal cardiovascular physiology are deserving of some attention also. Hypotension occurring during operation is a feature which demands close scrutiny by the anesthetist. Prompt and proper diagnosis, along with logical therapy, is required if acute and oftentimes

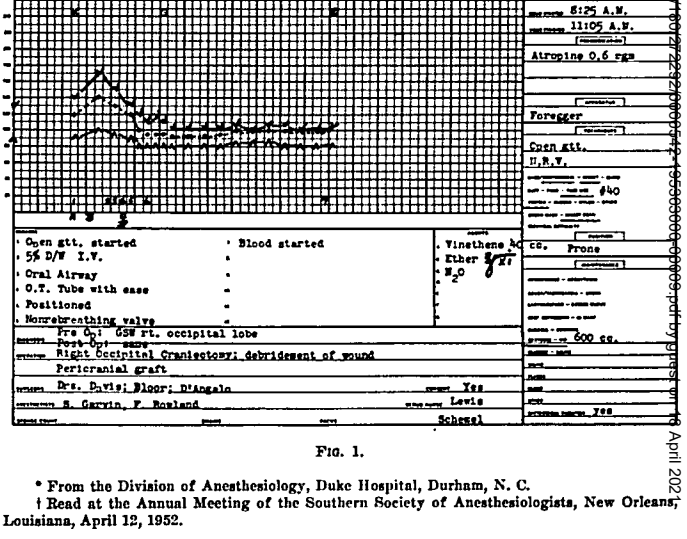

FIG. 1.

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disastrous emergencies, such as cardiovascular collapse, are to be prevented.

To obtain a proper perspective, it should be recalled that the five factors which combine to maintain the normal arterial pressure (4) are: (1) pumping action of the heart; (2) peripheral resistance; (3) quantity of blood in the arterial system, (4) viscosity of the blood and (5) elasticity of the arterial walls. During the altered metabolism of anesthesia and the strains and stresses of surgical procedures, any one or a combination of these factors may be disturbed to such an extent that hypotension develops. Sometimes the cause is obvious, but again

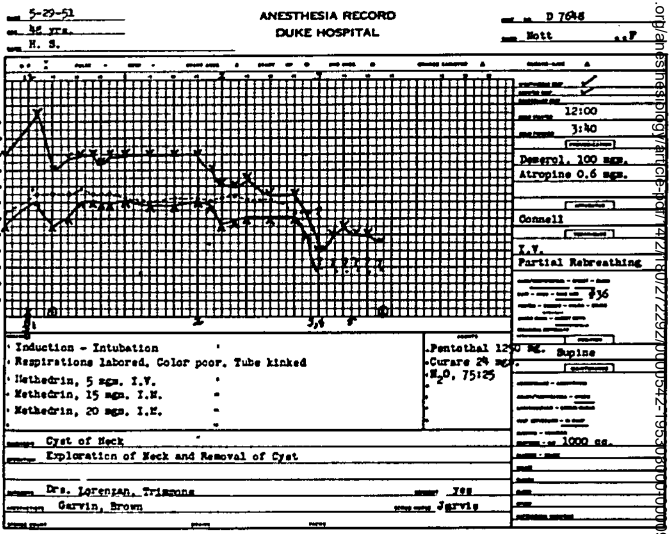

FIG. 2.

the etiology may be shrouded in obscurity. To the man at the head of the table the diagnosis and treatment are challenges which may tax his physiologic and pharmacologic background.

What exactly is meant by hypotension? At best it is a relative term, which connotes that the arterial pressure of a patient has fallen considerably from his recognized normal. How excited should one become over the development of low arterial pressure? The answer to that is very much of an individual problem, which involves the underlying physical status of the patient, and the spot analysis of the situa-

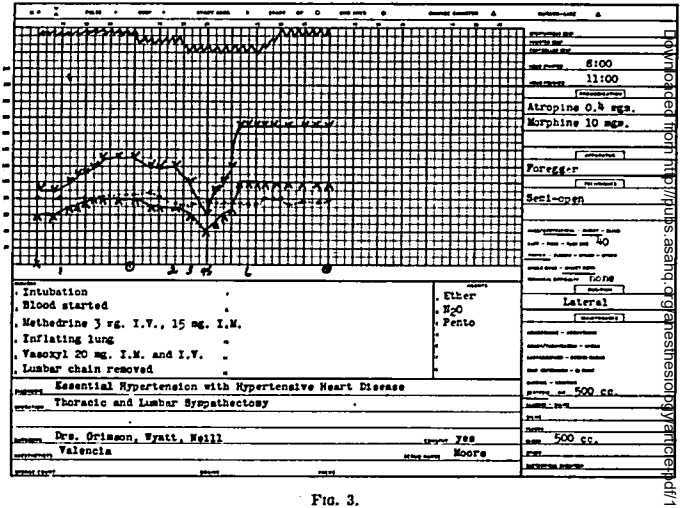

FIG. 3.

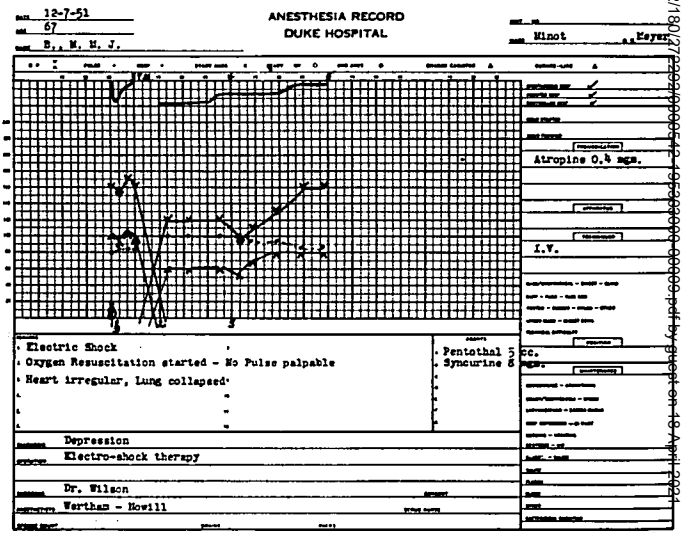

FIG. 4.

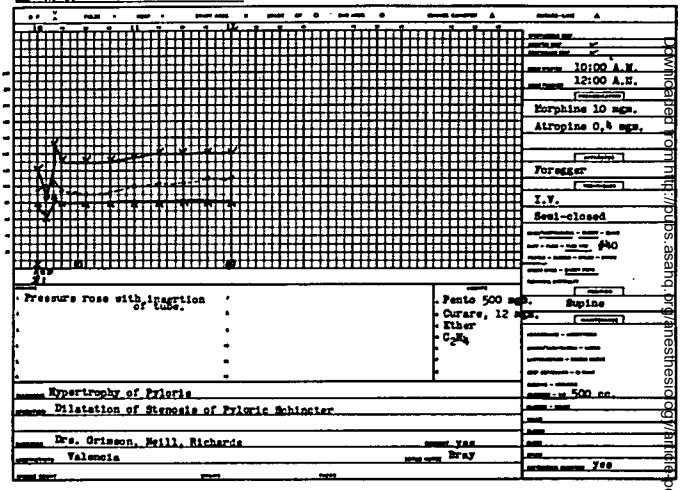

FIG. 5.

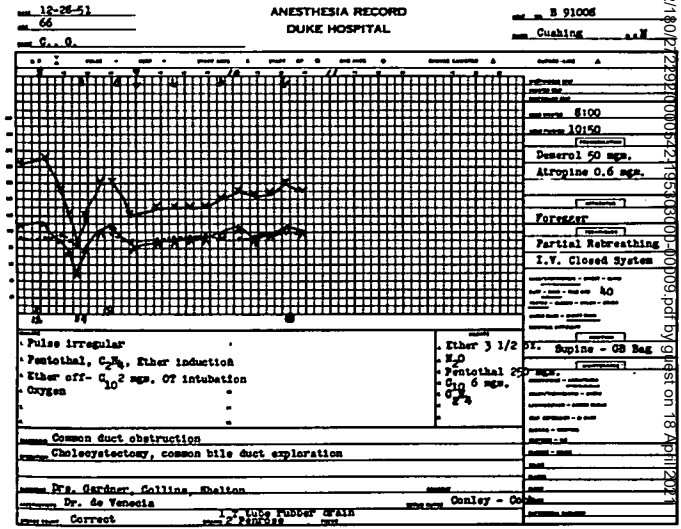

FIG. 6.

tion by the anesthetist. The patient represented in figure 1 had a preoperative hypertension of 200 mm. systolic and 110 mm. diastolic. After a quiet night, and prior to anesthesia, it was 130 mm. systolic and 90 mm. diastolic. During most of his operative course it was maintained at 100 mm. systolic and 80 mm. diastolic. Because his general appearance was good, airway clear, respirations adequate and anesthesia in a light plane, we were not worried. On the other hand, the patient reflected in figure 2 created great concern. His preanesthetic pressure was 220 mm. systolic and 130 mm. diastolic, and he was a known, poorly con-

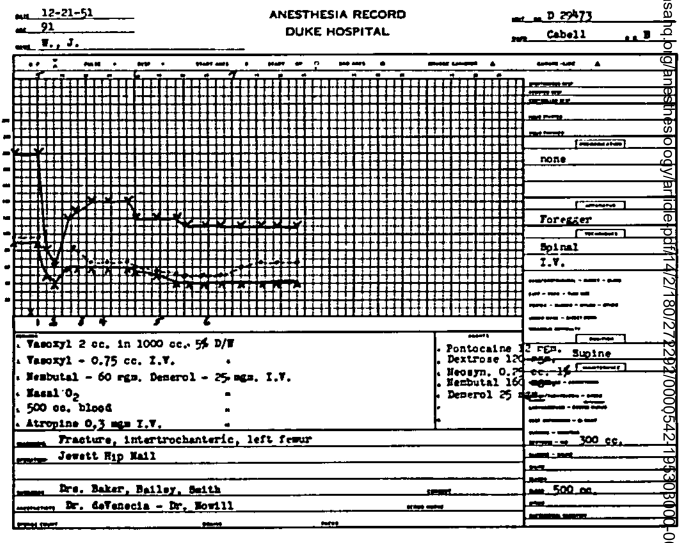

FIG. 7.

trolled diabetic. The first part of his anesthesia record shows a blood pressure course which tended to be maintained. However, his color became unsatisfactory and his respirations appeared labored. After two hours, it was discovered that the endotracheal tube was kinked in the posterior pharynx. The patient's color improved after rectifying this situation. Toward the end of the procedure, which involved minimal blood loss, there was a rapid fall in pressure to 80 mm. systolic; the diastolic pressure could not be determined. In view of the previous anoxia this was alarming. Anesthesia was in a light plane, oxygenation was adequate, but he showed little response to administration of

vasopressors. The worst fears were realized when an electrocardiogram after operation showed evidence of a massive infarct, not present before operation. These 2 cases emphasize that the development of hypotension presents a diagnostic problem which must be considered specifically in each patient.

Probably the most common cause of hypotension in the operating room is hemorrhage. When this is sudden and massive, the therapy is obvious—rapid transfusion with blood or blood expanders, such as dextran or polyvinylpyrrolidone. Vasopressors should be employed

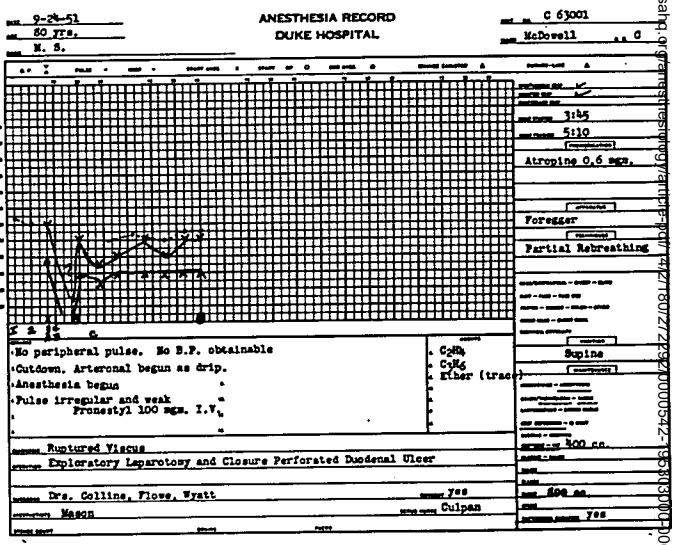

FIG. 8.

only to tide over an emergency; otherwise they may contribute to a false sense of security. Lightening the plane of anesthesia is important, as a patient in shock is also half-anesthetized. If the bleeding is slow but continual, it may act as a subversive element and not show the extent of its corroding influence for some time. One way of keeping abreast of the situation is to weigh the surgical sponges as they are discarded.

Evidence is being accumulated that many patients, particularly in the "old age" group, are coming to operation with blood volumes less than normal. When unavoidable bleeding puts a further drain on this

factor, and when anesthesia perhaps reduces the peripheral resistance the extra factors are added which diminish the circulating volume to shock levels much sooner than expected. These are the patients who, for want of better knowledge, are said to withstand operative interference poorly.

Of the several factors which may produce hypotension, the influence of premedicant drugs sometimes is overlooked. A moderate fall in arterial tension is anticipated from the quieting physical and psychic influence of the sedation, but in some patients, particularly those with

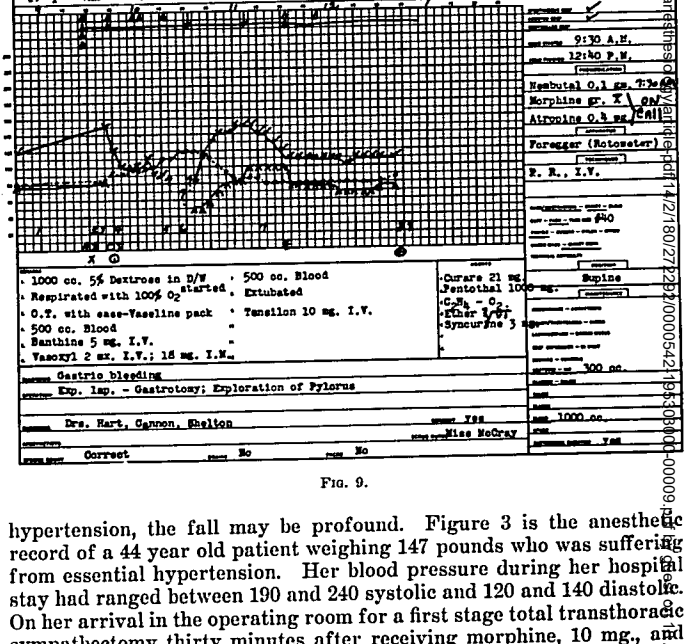

FIG. 9.

hypertension, the fall may be profound. Figure 3 is the anesthetic record of a 44 year old patient weighing 147 pounds who was suffering from essential hypertension. Her blood pressure during her hospital stay had ranged between 190 and 240 systolic and 120 and 140 diastolic. On her arrival in the operating room for a first stage total transthoracic sympathectomy thirty minutes after receiving morphine, 10 mg., and atropine, 0.4 mg., her blood pressure was 90 mm. systolic and 60 mm. diastolic. She was asleep and reacted only to strong stimuli. This situation confounded both surgeon and anesthetist. A course of masterful inactivity was elected, and during the next hour the pressure rose

gradually to 130 mm. systolic and 80 mm. diastolic. At that time anesthesia was deepened and the operation performed without untoward incident. This patient exemplifies the results of relative overdosage with premedicants. A modified form of this reaction probably occurs often, and may be masked by the induction of general anesthesia, with the blame attached to other factors.

The degree to which the anathema of the anesthetist, anoxia, may decrease the pumping action of the heart or hasten peripheral collapse is difficult to evaluate in most instances. A clear-cut illustration was

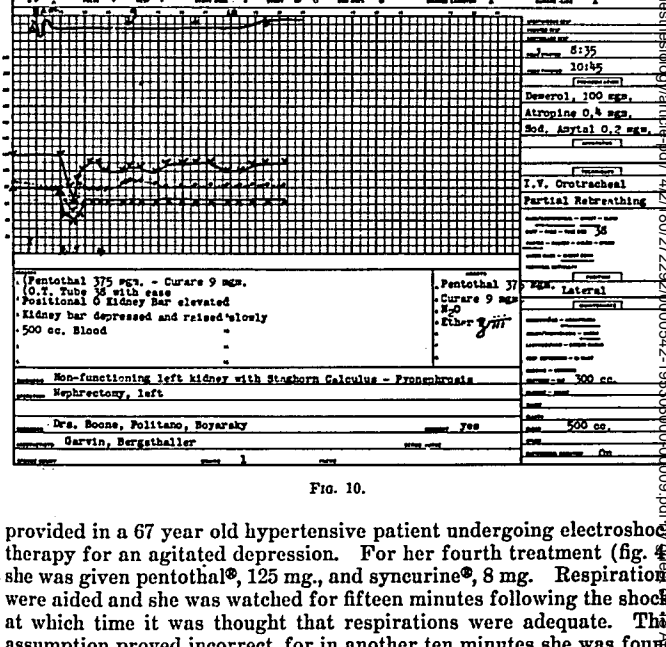

FIG. 10.

provided in a 67 year old hypertensive patient undergoing electroshock therapy for an agitated depression. For her fourth treatment (fig. 4) she was given pentothal®, 125 mg., and syncurine®, 8 mg. Respirations were aided and she was watched for fifteen minutes following the shock, at which time it was thought that respirations were adequate. This assumption proved incorrect, for in another ten minutes she was found to be cyanotic and without a palpable pulse or recognizable blood pressure. Artificial respiration with bag and mask and 100 per cent oxygen was begun immediately. Color returned slowly and in five minutes her pressure could be heard at 120 mm. systolic and 60 mm. diastolic. Aus-

cultation revealed a massive collapse of the left lung, and so an endotracheal tube was passed. Alternate aspiration of the tracheobronchial tree and artificial respiration, followed later by assisted respirations, restored this patient to an apparently normal status in about one and a half hours. The deleterious influence of lack of oxygen on the myocardium and the tone of peripheral vessels is well known, but seldom illustrated in so striking a manner. The possible role of anoxia should be considered in each instance of hypotension.

The rapid introduction of anesthetic drugs into the blood stream

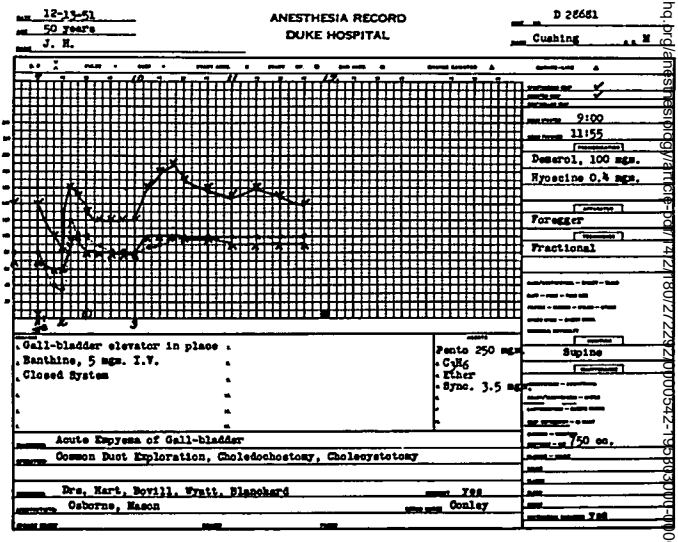

FIG. 11.

may produce specific effects on the cardiovascular system. The "shot gun" technic of pentothal-curare induction, with rapid intubation, is practiced commonly, but in many instances, if the physician is watching for it, produces a drop in blood pressure (fig. 5). Often normal tension is restored with insertion of the endotracheal tube. It is believed that ultra short-acting barbiturates have a direct weakening effect on the myocardium, which reduces cardiac output (5, 6). This effect can be demonstrated also in patients who receive large doses of barbiturates over a considerable period of time. After an hour or two of anesthesia,

definite narrowing of the pulse pressure can be observed, associated with an increase in the pulse rate (7).

Rapid induction with ether anesthesia is known to produce hypotension in some patients (fig. 6). This is believed to be the result of the uncompensated peripheral vasodilatation which occurs. If anesthesia is lightened, or as "autocompensation" develops, the arterial tension returns toward normal.

In like manner cyclopropane, rapidly introduced, may cause a hypotension associated with a bradycardia in some patients. This is typical

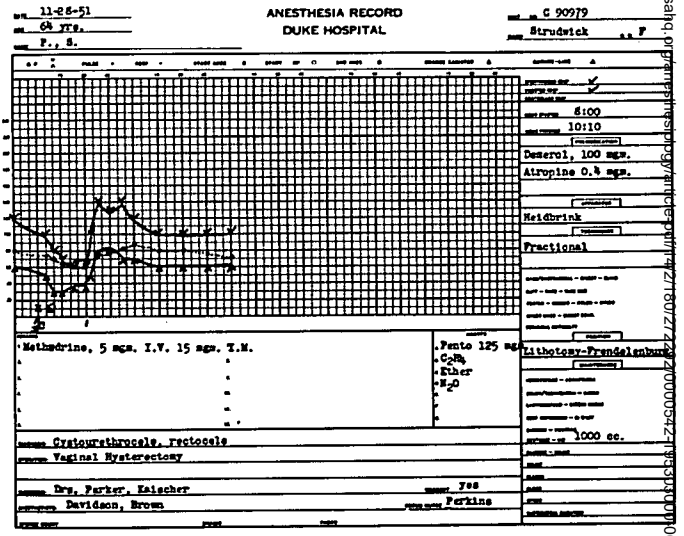

FIG. 12.

of an increase in vagal tone, and will be discussed later. Any anesthetic drug which is administered to the point of intoxication will markedly reduce blood pressure as a result of its central medullary depressant actions.

Concepts of the importance of the autonomic nervous system in anesthesia have been undergoing radical alterations in recent years. Part of this springs from the lighter planes of anesthesia employed in balanced narcosis, with the consequent functional preservation of many reflex pathways during operation. It is a fact that the anesthetist

today is confronted with many patient variables which can be traced ultimately to abnormal function of the autonomic system. Why should such diversified types of reactions be seen in surgical patients under anesthesia? Two hypotheses may be cited to explain the variability in type or degree of response by the patient: (1) In each individual there exists a balance between the sympathetic and the parasympathetic systems. In most patients one system tends to dominate over the other and thus the patient is said to be sympathotonic or parasympathotonic. (2) The autonomic system of the conscious person constantly is adapt-

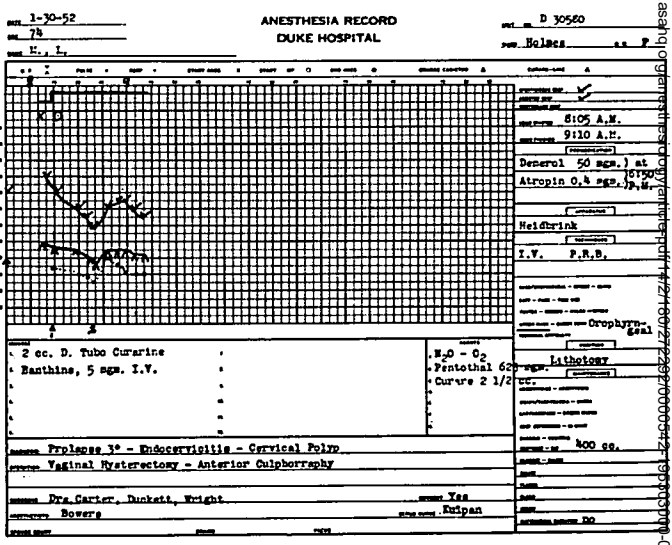

FIG. 13.

ing to or compensating for stresses placed upon it. The anesthetized patient to some extent has lost this capacity, and thus the burden of maintaining the environment is placed upon the anesthetist.

With this background, some of the episodes of hypotension which can be traced to the autonomic system will be reviewed. Sympathetic inactivity may result in a number of ways. Most dramatically this may follow spinal analgesia, as is shown graphically in the record of a 91 year old woman who fractured her hip (fig. 7). The sympathetic blockade in this instance was compensated for by the continuous administration of a vasopressor. The persistent relative overactivity of the para-

sympathetic system, as reflected in the bradycardia, was altered by administration of atropine. Another example of sympathetic incompetence is shown in figure 8, which is the record of an 80 year old diabetic, arteriosclerotic woman who was suspected of having a rupture of the sigmoid following an enema. On her arrival in the operating room blood pressure and peripheral pulse were unobtainable. The "traumatic shock" was not thought to be due to blood loss, and a continuous drip of arteronal (levophed®) was begun. When the pressure had risen to 120 mm. systolic and 78 mm. diastolic, anesthesia was in-

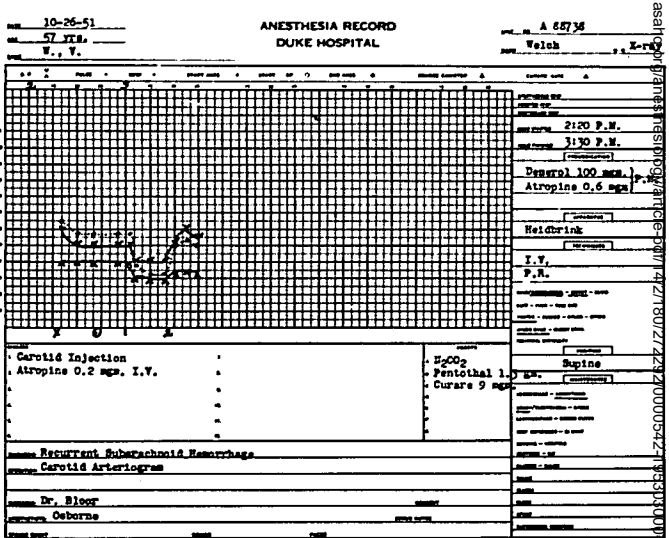

FIG. 14.

duced with ethylene and cyclopropane. As the operation proceeded the blood pressure level was maintained by administration of arteronal and whole blood. She was found to have a ruptured gastric ulcer.

Excessive stimulation of the sympathetic nerves may occur during abdominal exploration, and this occurrence has come to be known as the celiac plexus reflex. A rapid fall in pressure occurs without much change in pulse rate, as is seen in figure 9. Presumably, these potent reflex impulses cause pooling in the vast splanchnic area. Parasympathetic blocking agents, such as banthine®, have no effect in these patients, but a vasopressor restores the integrity of the pressure system

quickly. Burstein described this phenomenon as due to untoward activity of the splanchnic sympathetic nerves, and believed that it can be rectified indirectly by administering a parasympathetic stimulant such as physostigmine (8).

Reflexes which stimulate the activity of the parasympathetic system are most prone to occur during anesthesia. Such reflexes are reflected in the cardiovascular system by a pronounced fall in blood pressure associated with bradycardia. The etiologic factors involved are many, and only a few will be illustrated here. There are two or three phar-

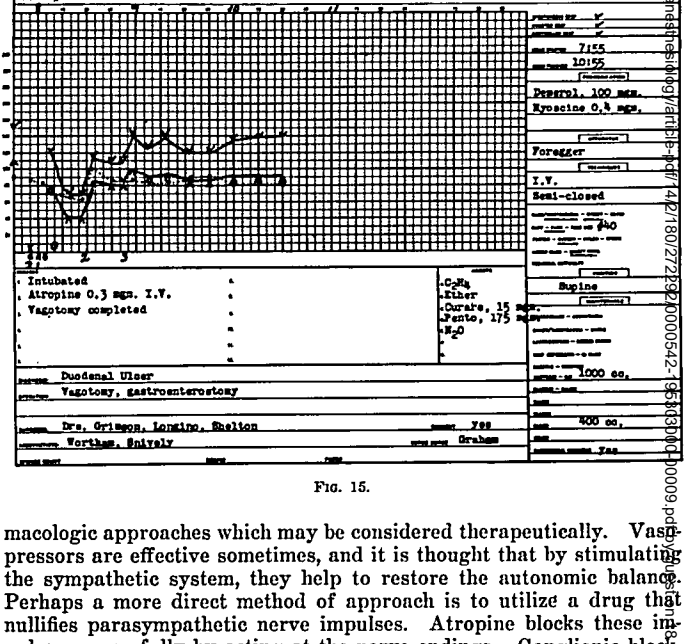

FIG. 15.

macologic approaches which may be considered therapeutically. Vasopressors are effective sometimes, and it is thought that by stimulating the sympathetic system, they help to restore the autonomic balance. Perhaps a more direct method of approach is to utilize a drug that nullifies parasympathetic nerve impulses. Atropine blocks these impulses successfully by acting at the nerve endings. Ganglionic blocking agents such as dibenamine® have been employed but they have the disadvantage of blocking both sympathetic and parasympathetic impulses. In the surgical patient such complete blockage might prove dangerous. More recently banthine, a ganglionic blocking agent which,

however, has highly selective blocking action on parasympathetic impulses (9), has proved useful in clinical practice in small dosage. Finally, evidence has accumulated to suggest that some muscle relaxant drugs reduce the reflex tonus of the vagus nerve. *D*-tubocurarine chloride is said to do this to some extent (10). The synthetic compound, flaxedil®, has a profound depressant effect on vagal impulses, to such an extent that tachycardia, indicating unopposed sympathetic action, usually follows its intravenous administration (11).

It is recognized that pharmacologic means are not necessary a

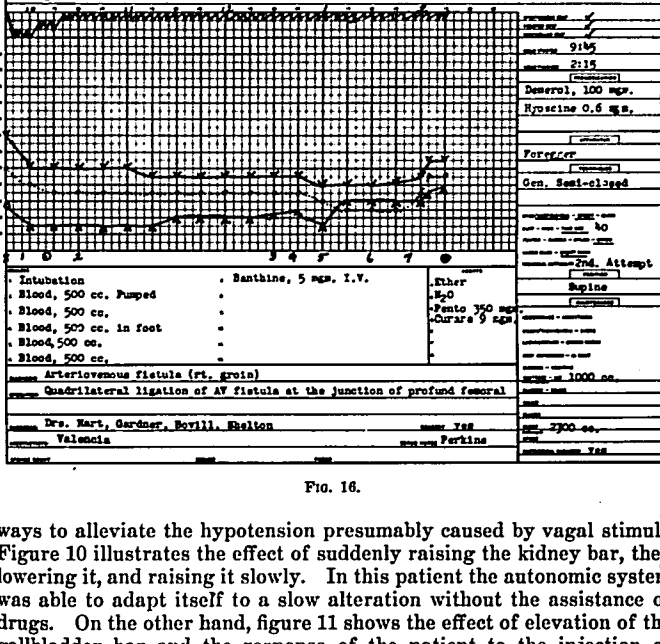

FIG. 16.

ways to alleviate the hypotension presumably caused by vagal stimuli. Figure 10 illustrates the effect of suddenly raising the kidney bar, then lowering it, and raising it slowly. In this patient the autonomic system was able to adapt itself to a slow alteration without the assistance of drugs. On the other hand, figure 11 shows the effect of elevation of the gallbladder bar and the response of the patient to the injection of banthine, 5 mg., intravenously.

The effect of vasopressors in counteracting abnormal vagal stimulation is seen in figure 12. Traction on the pelvic organs initiates potent pelvocardiac vagal reflexes producing a hypotension which responds well

to methedrine®. This same type of reflex can be alleviated equally well by intravenous administration of banthine, as is seen in figure 13.

Injection of dye into the carotid artery for arteriograms may initiate strong vagal cardiac impulses, as is exemplified in figure 14. In this patient normal conditions were restored by the intravenous injection of atropine, 0.2 mg.

The next chart (fig. 15) shows the vagal response which can occur with traction on the stomach. In this instance the hypotension was relieved by atropine, 0.3 mg., intravenously. It is interesting to note the further rise in pressure which coincided with the completion of vagotomy.

A reflex response presumably mediated by the vagus nerve to the heart has been noted during surgical repair of arteriovenous fistulas. This is initiated probably by the increased pressure in the carotid sinus or aortic arch. Figure 16 exemplifies the gradual fall in pressure and bradycardia seen in such cases, and the rapid response to injection of banthine, 5 mg., intravenously.

CONCLUSION

Hypotension during operation may be precipitated by numerous factors. It is important to assess the role of the possible etiologic influences, and to apply correct remedial measures as soon as possible. It is impossible to know when serious consequences may follow the sudden appearance of a profound fall in blood pressure.

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