might be responsible for capillary stagnation, reduction in blood volume, decrease in venous return, and the like required many experimental studies. . . . The bulk of evidence indicated that extracts or autolysates of most organs of the body, including breakdown of red cells, cause a pronounced fall in blood pressure. Admittedly, such experiments are far from crucial evidence that similar toxic agents are absorbed and produce shock. . . . Failure as yet to identify a toxic factor does not preclude its existence. . . .

"This very incomplete analysis of the purpose of many experimental studies should suffice to remove the charge that experimenters have unwittingly chosen unnatural experimental methods for producing shock. On the contrary, such experimental work has given rise to the conception that shock is induced by a vicious cycle of events. . . . It is not so strange that shock can be produced by catastrophes short of being immediately fatal; it is, on the contrary, remarkable that, so often, the body has sufficient resistance to withstand them. Unquestionably, greater emphasis on factors which prevent the development of irreversible shock rather than on those responsible for its development may supply a valuable key to our understanding of the problem. A careful study of the literature on shock and some experimental experience convince me that shock begins with a decrease in effective circulatory volume, as generally believed, but that the operation of some precipitating factor—or failure of some compensating mechanism—is required to cause the irreversible state of circulatory failure to which the term shock ought perhaps be restricted. If such a precipitating factor exists it is probably associated either (a) with creation of an irreversible damage to capillaries so that they no longer hold fluid or (b) with inability of compensating mechanisms to maintain an arterial pressure sufficient to supply vital tissues with blood. Both may be concerned. . . .

"It is, however, also conceivable and without prejudice to the view that capillary stagnation and loss of fluid represent initiating and sustaining factors in the evolution of shock, that the eventual determinant of circulatory recovery or failure does not lie in the capillaries at all. . . . Determinants of irreversible failure may concern (1) failure of moderator reflexes which normally act to stabilize blood pressures, (2) failure of mechanisms which mobilize blood from various blood depots into the general circulation, (3) failure of processes which aid venous return or (4) those which adapt the size of the large arteries to the cardiac output and blood volume. Finally, it is not impossible that the myocardium becomes impaired as a result of prolonged hypotension with the result that the heart fails to eject as much blood as it should with venous pressures that do exist." 19 references.

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"Originally the anesthetist's job was merely to keep the patient from feeling pain. Next it included helping the surgeon by relaxing the musculature. Only recently has the third duty come prominently to our attention, namely, to look out for the oxygen needs of the patient. . . . We have been hearing a great deal about 'ether convulsions.' The most recent opinions are that they are due to anoxia. . . . Anoxia or lack of oxygen in the blood and tissues may be acute enough to cause sudden death, or the cerebral changes first described by Courville, or dilatation of the heart, which can be demonstrated by x-ray. Anoxia may also cause complications of surgery which may not be blamed on the anesthetic in any way. Among
these complications are shock, circulatory failure, atelectasis and postoperative pneumonia, liver degeneration, renal failure, thrombosis and embolism. There are other causes for these conditions but anoxia plays its part. . . . The decreased blood pressure of spinal anesthesia is self-limited provided the patient is given oxygen so that anoxia does not complicate the picture. Shock is not a self-limited disorder but a vicious circle which readily becomes irreversible. . . . Anoxia in time causes failure of the vasomotor system but this vasomotor failure is the result, not the cause of shock. Vasomotor failure further expands the stream bed and makes the circulating blood volume even less adequate. . . . Anoxia causes the respiratory center to become less sensitive to normal physiologic impulses, breathing stops and the cardiac center fails only a little later.

"The anesthetist's part in this picture is to avoid those factors which contribute to the early stages of shock. It is the anesthetist's duty to avoid prolonged deep anesthesia and anoxia, and to detect early changes of shock and take appropriate measures. . . . It will be noted that I have not mentioned stimulants other than oxygen. . . . As a temporary expedient I do use neosynephrin, but less than formerly and I am critically examining results. Ample oxygen delivered to the lungs by a free airway will do much to prevent the development of shock and is basic to the treatment of shock along with the provision of adequate circulating blood volume. . . . The patient who enters the hospital on the morning of his operation does not take as smooth an anesthetic as the patient who has been hospitalized for at least one night. It is also helpful for the patient to make the acquaintance of the anesthetist before the time of the operation. . . . We often give the preoperative hypodermic too late for it to be fully effective. . . . Scopolamine supplements morphine better than does atropine. . . . The preliminary sedation should be prescribed with the agent and kind of anesthesia in mind. . . . Doubtless we should use oxygen postoperatively much more frequently than we do. . . . Lung abscesses can readily be caused by inattention on the part of the anesthetist. The giving of a general anesthetic to a patient whose stomach is not empty is a dangerous procedure, but one that is sometimes necessary. . . . Liver damage may be caused by certain anesthetic agents, notably chloroform and trichlorethylene, and to a less extent, ether and avertin. Liver degeneration is much more likely to occur in an obese individual or one with low liver glycogen; it is sure to occur with prolonged anoxia. . . .

"I believe we are soon going to have excellent new aids in the prevention of thrombosis and embolism and that they will be along the line of increasing the clotting time of the blood in selected cases where those circulatory complications are most to be feared. . . . One should be very careful to provide a free airway for the edentulous patient. Teeth prop the mouth open and help maintain an airway and unobstructed pharynx, especially when a simple airway is placed between them. A rubber airway is much preferable to a wire one since it can be slipped out if the patient is waking and bites on the airway. . . . I believe that avertin, like spinal anesthesia, is safe only if oxygen is readily available to take care of the occasional severe reaction. . . . Symptoms of oxygen lack and carbon dioxide increase may include any of the following: alterations in the pulse, dilated fixed pupil, dry cornea, depressed or irregular or sighing respiration, raised systolic pressure, changes in pulse pressure, muscle twitchings or convulsions, cyanotic or ashen color, anxiety, restlessness, delirium, air
hunger, vomiting and retching, increase of moisture in the lungs. The prevention consists of intelligent preoperative care of the patient, the individualization of preoperative sedation, the use of airways, the support of the patient's chin (with a finger constantly on the pulse) and constant minute watchfulness. The blood pressure, pulse and respirations should be followed and recorded at the time and not guessed at and written down after the operation. It is hazardous to give any general anesthetic with a closed system of complete rebreathing from a bag without knowledge of the patient's blood pressure. It is also inviting trouble that may make the surgeon's best efforts of no avail for the anesthetist to give even open drop ether to a poor risk patient or to any patient for a very serious procedure without constant watchfulness and interpretation of the blood pressure." 5 references.

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"The consumption of oxygen on the Surgical Services at the University Hospitals [State of Iowa] comprises approximately 50 per cent of the total amount used. The indication for oxygen therapy is the same as under any other circumstances, namely, the correction of oxygen want. Specifically, the oxygen is a valuable adjunct in the treatment of shock whether it be traumatic shock, as seen in an accident case, or surgical shock developing either on the table or immediately postoperatively. Any patient in shock is suffering from stagnant hypoxia and is in oxygen want because of the failure of the sluggish circulation to supply enough oxygen to the tissues, hence, the rapid, thready pulse; the shallow, rapid respirations and the pale or subeyanotic appearance so often seen. It has been observed that if oxygen therapy is instituted along with other measures taken to combat shock in the surgical patient, such as fluids, blood, heat and shock position, recovery from the shock is facilitated. In fact, certain cases have failed to respond as rapidly as they should to the supportive treatment until oxygen was added.

"The toxic thyroid patient with his coincident increased metabolism requires excess oxygen and is a frequent user of oxygen therapy on the surgical service. The tent is of benefit to these individuals because of the cooling effect in addition to the oxygen. The thoracoplasty patient needs oxygen not only to help overcome the shocking effects of the actual surgical procedure, but also to maintain normal arterial oxygen saturation until he can compensate for the effects of the surgically induced pulmonary collapse. The same holds true for the patient who has had a lobectomy or pneumonectomy. The neurosurgical patient needs oxygen postoperatively to help combat both the surgical shock and the respiratory depression often connected with such procedures. It is felt that the administration of high concentrations of oxygen in conjunction with the use of the Wangensteen suction or Miller-Abbott tube helps in reducing the severity of postoperative distention in the abdominal cases. The mask is of particular benefit for this type of therapy. Needless to say oxygen therapy should be instituted at once in cases of postoperative pulmonary complications such as atelectasis, bronchopneumonia, lobar pneumonia or pulmonary edema. Another type of case requiring oxygen is the patient suffering from a fulminating infection . . . It has been stated in recent literature that oxygen therapy also plays an important part in the proper healing of wounds." 4 references.

J. C. M. C.