public health boards, however, have immense clinical material, for they are in touch with a large number of physicians and they have machinery for the assembling of desirable data.

Our own experience in the latter respect bears directly on the question. We have found the medical profession of the state interested, in sympathy with our efforts, and most helpful in every way almost without exception. The families of the cases investigated have given us every possible assistance, and have welcomed the inquiry. The legislature has been most liberal, and has granted us in all \$20,000 in three separate years, without question. With a profession interested and sympathetic, a public ready to help, and a legislature ready to back such work financially, the Massachusetts experience points to the advisability of such concerted investigation and research between public health boards and the scientific departments of medical schools.

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SHOCK. A REVIEW OF THE THEORIES AND EXPERIMENTAL DATA TO DATE.

BY EDWARD H. RISLEY, M.D., BOSTON.

THE phenomenon known as shock was a condition recognized by careful observers as early as 1568, when Clowes, and later in 1719, Weismann, spoke of it as a condition probably caused by the presence of a foreign body in the wound or blood. A little later than this the condition was attributed to a disturbed molecular condition in the tissue.

From a purely historical point of view the literature devoted to shock may be divided into three periods:

(1) From 1795, when the word was first used as we now use it, down to the beginning of the era of experimentation in 1876.

(2) The period from 1870 to 1885, devoted to careful experimental investigation, and

(3) From the re-opening of the question by Crile in 1889, to the present time.

From 1885 to 1889 practically no papers, others than purely casuistic ones, appeared on the subject.

Nearly half a century before Crile's work, Keen, Mitchell and Moorehouse advanced the theory of vasomotor exhaustion, and experimental work by Fischer in 1870 seemed to confirm this theory, i.e. of a vasomotor paralysis with flooding of the splanchnic veins and contraction of the peripheral vascular system. This was the recognized phenomena but no adequate explanation of its physiology was at hand.

Brown-Sequard believed the condition to be one of central anemia due to persistent contraction of the capillaries throughout the vasomotor centers.

Moulin: A reflex paralysis in the strictest sense of the word; a reflex inhibition, probably affecting all the functions of the nervous system and not being limited to the heart and blood vessels only.

Boise: Hyperirritation of the entire sympathetic system with resulting stimulation of vasomotor nerves, contraction of arterioles and spasmodic heart action.

Kinneman emphasizes the rôle of a fall in temperature as the principal causative factor in shock, and as a result of extensive animal experimentation, finds that shock must not be considered as due to the lowering or exhausting of any one bodily function, but as a composite condition embracing an interference with normal height of the blood-pressure, interference with the respiratory act and a marked fall in body temperature. Of these, as shock progresses, the most uniform and progressive factor is the fall in temperature. Dogs, after shock, were immersed in a bath of saline at body temperature, and others in a bath of higher temperature. These experiments showed (a) that a sufficient fall in body temperature can cause a decrease in respiratory rate and a marked fall in blood-pressure, which in itself constitutes a shock; (b) conversely, a limiting of the fall of temperature limits the fall in pressure and prevents a fall in respiration. (c) On the other hand, a rise in temperature causes a rise in blood pressure and respiratory rate, reduced by shock, with the result of a gradual annihilation of all symptoms. Thus, of the three factors, temperature commands first place by its power of production, of limitation and amelioration of the composite condition—shock.

As the problem stands today there are several contradictory theories pretty well based on rather extensive experimental evidence. It is to the proper interpretation of this accumulated experimental data that we must look for the physiologic cause rather than to the theory or opinion of any one worker. It is interesting, and at the same time puzzling, to note that each observer is equally sure that he has discovered, through experimentation, the fundamental or chief underlying factor. Thus Crile maintains that vasomotor exhaustion is the primary cause. Boise states that cardiac exhaustion is the prime factor, whereas Howell believes that both cardiac

and vascular changes are at fault; Kinneman (as quoted above) stands for a disturbance of thermogenetic functions; Henderson for the reduction of carbon dioxid content of the blood and tissues (acapnia). Meltzer from experimental evidence, disagrees with Crile, stating that instead of vasomotor exhaustion there is reflex inhibition of the activity of the vasomotor center of the cord. In regard to this particular question it is very probable that personal equation plays a large rôle and that what Meltzer calls the initial symptoms or rapid onset, Crile does not recognize till fully developed shock is evident, (the slow onset).

What is exactly meant by shock is also hard to define. The word represents an intricate symptom-complex and should not be understood to stand for a specific clinical actuality. For this reason we can see how divergent certain views as to its underlying factors may be.

It seems best here to give a synopsis of the work of these men who have recently gone most thoroughly into this subject and then later to correlate these results as far as possible. I shall reserve Crile's work till last as his is the most extensive and seems to bring the subject more nearly up to date.

If we think of shock as a condition induced by fear, exposure, infections or trauma, in which there is exhaustion of nerve cells, principally those of the vasomotor centers but also, probably to a much less extent, of other centers in the medulla, such as respiratory and cardiac, and consequently lowered vasomotor tone and cardiac and respiratory depression of so even a degree as often to result in death, we have before us the general picture, the physiologic cause of which these experimental evidences are trying to discover.

Malcolm of London, dealing with the problem from a purely clinical point of view, believes that if vasomotor exhaustion is present, then the innervation of small peripheral arterioles, being cut off from central control, must be relaxed. This should result in an over-filling of the peripheral vessels; but as a matter of fact, all clinical evidence speaks against such over-filling. The body surface is cold and pale, the mucous membrane anemic, incisions made during shock are almost bloodless. Crile answers this objection by stating that the state of relaxation of peripheral blood-pressure is perfectly consistent with such a condition as the blood has already flowed *through* the dilated arterioles and has collected in the deep veins where it is demonstrable at death.

Bartlett shows that the effect of removal of blood from the body in hemorrhage is not identical with that accompanying a fall of blood-pressure due to trauma. It would seem that the vasoconstriction following bleeding indicates an automatic protective mechanism which is not evident in pure shock. This author believes, with Crile, that the decreased blood-pressure

following trauma is due to decreased vasomotor tone, but does not state whether he considers this a complete exhaustion or not.

Leibig and Lyon, however, working only on vagi by electrical stimulation prove that vasomotor exhaustion does *not* occur in shock but rather vasoconstriction. This work is limited to this one class of experiments and does not prove alike for all kinds of surgical trauma and shock.

Brown ascribes the difference between the sudden shock, often seen on the operating table, and the shock of more insidious onset, seen hours later, as follows: The shock noticed during operation is due to the combined effect of the anesthetic in lowering the blood-pressure and the temporary exhaustion of the vasomotor centre by repeated stimulation of the partially narcotized nerve cells together with hemorrhage of varying degree, still further lowering blood-pressure and vagus inhibition of the heart. The later phase of shock depends on exhaustion of the food supply stored up in the vasomotor cells. Owing to the fullness of the circulation through the centers due to the depressed blood pressure, consequent on the exhaustion, no further supply of food is taken up and a vicious circle is set up, causing further exhaustion and shock. This theory has a definite experimental and clinical basis and furthermore seems reasonable.

We now come to a more recent and more radical and widely different view of the underlying principles of shock, but one also well established by experimental evidence—that of Henderson.

This author believes that the underlying cause is insufficiency of the carbon dioxid content of the blood. The tension of this gas in the circulation is the normal stimulus of the respiratory centre, and consequently is regulated automatically in a healthy individual under normal conditions. If, however, the rate of respiration be markedly increased the over-ventilation of the lungs causes an increased inspiration of oxygen resulting in a condition of over-oxygenation of the blood with diminished carbon dioxid or—acapnia. The respiratory centre is thus robbed of its normal stimulus and spontaneous respiration ceases, although the heart continues to beat (apnea). The sensitiveness of the respiratory centre for carbon dioxid varies. If this sensitiveness is acute and the acapnia is mild in degree a vigorous circulation will permit vital processes to continue long enough for the re-accumulation of sufficient carbon dioxid in the blood to restore spontaneous respiration. If, however, the acapnia is intense, apnea continues and death takes place from oxygen starvation of the heart. With acapnia there comes dilatation of the splanchnic veins and a fall in blood pressure. The tonicity of the walls of the blood vessels is in direct proportion to the carbon dioxid content of the blood. Henderson quotes Crile to show that in his (Crile's) experimental

work it was necessary to resort to artificial respiration in order to keep the animal alive long enough to procure the evidence of the vasomotor origin of shock. He, therefore, concludes that the respiratory failure is the primary change in shock and that the other phenomena are of secondary origin.

Henderson advocates the use of oxygen and carbon dioxid 20 to 1 by inhalation in treatment of shock, even by intra-tracheal insufflation if necessary or rebreathing with a paper bag to renew the stimulating effect of CO_2 .

(N. B. This has been recently tried with success and reported by Cotton in three cases, when shock seemed to be due to excessive respiration; one a sobbing, hysterical woman.)

If apnea is prolonged, asphyxial acidosis develops. If the acapnia is not too intense these products of incomplete tissue combustion induce isolated gasps, followed by Cheyne-Stokes breathing and prevent immediate death. After intense bodily suffering failure of respiration is the usual form of death. It is only when the pain is sufficiently continuous to prevent apnea that the slower process of failure of circulation develops.

It seems to the writer that Henderson's theory is sound as far as it goes, his work convincing and his reasoning logical, but that he does not include, in regarding deficiency of carbon dioxid, all the features of shock or the fundamentals present at the very onset of the condition.

Shock is a complex condition, not a definite symptom, but a symptom-complex, dependent on more than one causative factor.

Because of the length of time over which he has carried his experimental work and the thoroughness and extent of his researches, Crile's work seems more than any other to deserve attention and recognition, and because he has built up a well founded line of treatment based on his experimental work it seems consistent that more reliance should be put on his theories and observations and results than on those doing only fragmentary work on separate phases of this complex problem.

It is well known that severe infection, hemorrhage, exhaustion from physical exertion, exophthalmic goitre, surgical trauma and overwhelming emotion are the common predisposing causes of surgical death. An operation that would present virtually no risk in the uncomplicated stage may readily prove fatal in any of these handicapped cases. The question to determine, therefore, is, "What are the factors in the technic of a surgical operation that may further impair the brain cells and thus turn the balance to a fatal termination?"

In shock, the essential phenomenon is a diminution of blood-pressure. This must be due to the exhaustion of the cardiac muscle, cardiac centre, blood vessels or vasomotor center. Experiment shows that it is not the cardiac muscle

or its controlling nervous mechanism that is at fault but a loss of peripheral resistance. Fatigue of blood vessels can be excluded also experimentally. It, therefore, must be the vasomotor centre which suffers injury and causes reflexly the symptoms characteristic of shock.

Quoting from a large series of experiments carried out on dogs under ether anesthesia, and under varying conditions of temperature, etc., it was found that (1) the duration of an operation alone was a potent factor in the production of shock. (2) Animals may be killed from continuous anesthesia alone, though this anesthetic be carefully administered over a long period. (3) There is strong evidence that chloroform, even barring accidents, is a more potent factor in destroying the animal than ether. (4) Exposure to air is a great factor in producing shock, reducing body temperature and causing drying of tissues. This is strikingly observed in case of the peritoneum, pleura and brain, and affects particularly the vasomotor mechanism. Exposure causes first a hyperexcitability, which is soon followed by non-excitability. Exposure of the peritoneum causes splanchnic vascular dilatation and also affects respiration unfavorably. (5) The element of time in all abdominal operations was unmistakable. There is sufficient evidence at hand to establish at least a high degree of probability that the shock in operations on the splanchnic area is largely caused by disturbance of the *local* splanchnic vasomotor mechanism.

The experiments of Mall show that the splanchnic nerves are vein-nerves and control this large splanchnic vascular area. Every experiment in this area gave evidence of the dilatation of the vessels controlled by these nerves and the decline of blood pressure occurring *pari passu* with this dilatation.

The great shock caused by injuries to the male genital organs is probably due largely to vasomotor dilatation of the splanchnic area. It is safe to say that the "excluded splanchnic circulation" experiments only prove the splanchnic vasomotor factor plays but a part even in such injuries as involve its own area alone; there is no evidence tending to show that in operations in areas of the body other than the splanchnic and genito-urinary tract, that the splanchnic factor plays a special part, probably not much more of a part than in every other area of like vascular capacity. Experiments in which this area was not involved did not show a condition of vascular distension in this area different from that of other areas.

The output of the heart is in direct ratio to the pressure of the vena cava, and not at all to the height of the aortic pressure. The nervous pressure then determines the heart's output. In the condition of profound shock the accelerated cardiac action is taken as evidence of the effort of the regulating centers to recover the lost blood-pressure, but the vasoconstrictor or pres-

sor mechanism is not active; it is probably paralyzed or seriously impaired. The splendid action of the heart under these conditions, on infusion of salt solution proves it to be well capable of further action. It is evident that the heart's action is the last of the great vital functions to fail. Post mortem appearances show the large venous trunks full, sometimes enormously engorged, the arteries empty, the veins of the splanchnic area not more distended than those of the somatic, unless the experiment had included some procedure in the splanchnic area. In a general way, it may be stated that in the organs and tissues, such as intestines, the experiments on which produced a primary fall in blood-pressure, shock was induced earlier than such tissues and organs as the extremities, which produced a primary rise in blood pressure. Gross tissue resistance proved to be a very fair index to resistance.

After shock has been fairly induced reflexes are never so acute, vasomotor actions were sluggish and ineffectual and when blood-pressure declined it was, as a rule, slowly or never regained.

Extensive experimentation shows, therefore, that in shock the essential factors to be considered are trauma, the anesthetic, a primary rise with a following fall in blood-pressure, decrease in body temperature and vasomotor inhibition, paralysis and then exhaustion. In addition to these physiologic changes more recent experimental work of Crile and his associates have shown another, and perhaps entirely unlooked for, physiologic and pathologic process to take place in the presence of shock. I refer to the recent work showing extensive cytologic changes in the brain, as a result of trauma, fear, infection, etc. Animals were overtransfused so that the factor of anemia of the brain was wholly excluded during the application of the shock producing trauma. In every instance, morphologic changes in the cells of all parts of the brain were found, especially in the cortex and cerebellum. It required more trauma to produce equal changes in animals protected against low blood-pressure than in those whose blood-pressure gradually declined during the experiment. The structural changes include changes in the absolute size of the cells, in the size relation of the nucleus, plasma, surrounding membrane of the cell, nucleus and the general contour of the cell. As to gross size, the increase varies from slight to even eight times the normal, the nucleus becomes relatively larger, the limiting membranes may be even ruptured, causing distortion of the cell. In extreme shock the cell completely loses its contour and presents the appearance of a disorganized mass of protoplasm.

At first, in shock, there may be increase in the reaction to Nissl stain, but later, as shock increases, the reaction becomes less and in the final stages there is little or no stain reaction. Even in cases of fatal shock, however, not all of the

cells are thus altered. On morphologic grounds, when the nucleus, nucleolus and the cell body become so broken down as to have an undifferentiated mass of protoplasm, the cell is almost certainly dead and will never regenerate, *i.e.* the brain cell is finally deprived of its discharging nervous energy and becomes exhausted just as exhaustion follows too prolonged muscular exertion. To use a vulgar word, the cell literally and figuratively becomes "busted." The gravity of a given case is well indicated by the percentage of cells that are thus damaged. It has been shown, likewise, that the cells of the brain in various diseases also experience the same changes as the one found in shock. A nerve cell that has been impaired by overwork, anemia, infection, drug poisoning, or Graves' disease, obeys the same general law of change. Therefore the changes that are seen in the brain cells are not in the least specific for any etiologic factor, for they appear to be common to all of the above named conditions so far studied.

These studies only serve to further illustrate more forcibly how complex a state shock is and how many factors enter into its physiology. These cytologic changes would rationally explain many of the post-operative neurasthenic states so often seen.

Crile goes further and states that there is strong negative evidence that traumatic impulses are not excluded by ether anesthesia from the part of the brain that is apparently asleep. For if this factor of fear be excluded and if, in addition, the traumatic impulses are prevented from reaching the brain by cocaine, then, despite the intensity or duration of the trauma, within the zone so blocked, there are found no morphologic changes in the brain cells. If, on the other hand, no blocking was used, cell changes are everywhere present. Therefore, although ether anesthesia produces unconsciousness, it apparently protects none of the brain cells against exhaustion from the trauma of the surgical procedure. Ether is, so to speak, but a veneer that covers the deeper suffering of the patient (or patient's brain cells).

Crile believes that ether alone is one of the determining factors in the production of shock. In a series of parallel experiments on animals with ether and with nitrous oxid it was found that the damage to brain cells under ether was four times greater than under nitrous oxid.

Having reviewed the factors on which Crile believes shock is based, we are now in a position to consider the logic of his solution of the problem of treatment. It seems to me it is an extremely logical and reasonable line of deduction. Some of it is old, but a great deal is certainly new and advanced, well-founded on experimental work and extremely efficient from a practical point. He adds practically nothing new to our routine of treatment in cases in which shock has already developed, *i.e.*, heat, rest, morphia, intravenous infusion, bandaging

of extremities, elevation of the foot of the bed and transfusion. He believes that intravenous infusion is of prime importance because it causes, in the first place, an increase in the venous pressure in the vena cava and consequently the output of the heart is at once increased, the amplitude of the strokes lengthened, the chambers being full, the contractions become more forcible and blood-pressure begins to rise. The combination of small, frequently repeated hypodermic injections of strychnia together with saline infusion produces a more constant pressure than ether alone. Over-stimulation is followed by greater depression, hence large doses of strychnia are absolutely contraindicated, for such doses produce vasomotor paralysis and shock the same as trauma does. It is as reasonable to treat shock with large doses of strychnia as to treat strychnine poisoning with trauma. No justification can be found, experimentally, for the use of alcohol, nitroglycerin or anylnitrite as stimulants. As regards alcohol, in not a single instance was there a sustained improvement in the blood-pressure or respiration. On the contrary, the most constant and marked effect on the blood-pressure was a decline. The final breakdown was more sudden with drugs than in control animals. Adrenalin, however, administered cautiously and continuously, with a limited amount of saline, proved the best stimulant, in addition to external pressure by Crile's rubber suit or bandaging of extremities.

The great advance made by Crile in the treatment of shock has been in elaborating a logical *prophylaxis*. This we are in a position to study and to use clearly his reasons for.

It is an unpleasant thought that although our patient is unconscious from general anesthesia, yet the nerve impulses inaugurated by operative injury of the nerves reach the brain and produce harmful changes there which are the precursors of shock. It is clear that if general anesthesia cannot prevent the damage done to the brain in the course of operation some means must be devised to prevent this damage. It is known that cocaine, in addition to its ability to block sensory impulses, also blocks impulses that cause the response of the entire nervous system, under general anesthesia. If we combine, therefore, a local with a general anesthetic and avoid fear previous to operation, by the use of sufficiently large doses of morphia to bring the patient to the operation in a quiet, contented frame of mind, it matters not how poor the risk or how extensive the operation, the nervous system is protected and the immediate operative risk is eliminated.

The elimination of the factor of fear is an extremely important one and can be entirely accomplished only by the most careful co-operation on the part of the nursing and operating staff. The patient should be told of all the preparations to safeguard his welfare before,

during and after operation; his room should be quiet and free from outside disturbances.

Fear is stronger than will, but under morphia a mental state is produced in which one is neither brave nor a coward, because this drug destroys the associational power of the brain and the patient is left in a quiet state of mental and physical repose.

If we can, therefore, prevent fear and obtain an innocuous substitute for ether, and block harmful nerve impulses from the brain, we have a condition best described by the word *anoci-association*, that is one in which the patient is both mentally and physically protected from nocuous impulses or influences.

(The difference between anoci-association and anesthesia is as follows: Although inhalation anesthesia confers the beneficent loss of consciousness and protects from pain, it does not prevent the nerve impulses from reaching and fatiguing the brain cells and hence does not prevent shock or the train of later nervous impairments so common following shock, i.e. neuros-thenic post-operative states.)

The *prophylaxis*, therefore, which is to produce a state of *anoci-association* consists first in gaining the confidence of the patient, reassurance of a favorable outcome, the generous enough use of morphia before operation to produce a state of mental relaxation and content, nitrous oxid and oxygen anesthesia, nerve-blocking by cocaine or novocaine, great care to prevent loss of body heat, injury to tissues handled, and trauma to any but the immediate site of operation, speed reasonable to safety and a similar anocuous after-care. Crile, in abdominal operations, uses the quinine and urea hydrochlorate injected into the peritoneal edge before sewing up. The anesthesia produced locally by this drug lasts for 24 to 48 hours and protects the patient almost entirely from the wound pain, as it is the pulling of peritoneum, which is the greatest factor in the causation of the post-operative wound pain.

During the past year the author has had the opportunity to personally follow the results of Crile's prophylactic treatment in a large number of cases treated in this way by Dr. Hugh Cabot, for whom he gave ether, and in several cases of his own. I was immediately struck with the fact that patients required about a third less ether (given by the drop method) than when not treated by anoci-association. The ether was also taken more easily. Nausea and vomiting were absent in a great many cases and slight in others. One patient of my own, who had been operated on (laparotomy) four times before and was well familiar with the post-operative discomforts, told me that "the absence of the apprehension of pain—and the absolute lack of backache and wound pain were the most striking things to him." He was very much surprised at the success of the method and in two subsequent laparotomies has had no fear

or dread and has had a very comfortable time.

No sepsis has resulted from the use of the urea compound as far as I know. Patients distinctly neurasthenic are the type of case in which the most noticeable results are obtained. It certainly is most gratifying to witness such absolute post-operative comfort. The surprise to the patient is even greater than to the surgeon. I cannot say enough in praise of this whole procedure, as I feel sure it adds a strong element of safety in any surgical operation—especially operations in the abdominal cavity and gives the patient a sense of comfort not obtainable in any other way.

Before closing this paper, it is necessary to give a summary of some recent work done in England by Gray and Parson. These authors worked along the same line as Crile, especially on blood-pressure. They divide shock into three stages:

(1) Stage of stimulation or pressor stage, which is characterized by a rise in blood-pressure.

(2) Stage of depression or depressor stage, characterized by a gradual fall in blood-pressure, and

(3) Stage of equilibrium, when the active, abnormal, afferent impulses are in abeyance.

These authors believe that one of the most important factors in the production of shock is the region of the body which is attacked. To each tissue, organ, structure or operation a certain "shock value" may be ascribed. Consequently shock values vary directly with the relation of pressor to depressor fibers in the nerve supply of a part. In general, those parts which are physiologically most active contain the largest proportion of depressor fibers in their nerve supply and show the highest shock value. These authors made extensive observations on humans and supplemented them by like operations on dogs. The literature is very extensive and full and abundantly illustrated by charts of blood-pressure. They definitely and emphatically state that the most careful histologic examination of the brains and spinal cords of shocked animals failed to reveal any changes in the motor cells as described by Crile and Dolly. In the spinal cords they found no cystolytic changes at all. The cerebral cortex showed very little change and the Purkinje's cells showed no striking alterations. They conclude that there is no fatigue in shock of nerve centers to be detected histologically. Sensory cells, however, show some slight damages which, however, are not constant. The authors conclude that shock cannot be accurately defined, for the term does not express a state or condition, but rather a mechanism, which establishes, first, a defense of the organism against the harmful effects of injury and next, the protection of the vital centers from a degree of exhaustion from which they could not recover, *i.e.* shock is the reaction of

the central nervous system to exaggerated or abnormal afferent impulses. The study of shock means the study of pathological vasomotor reflexes regarded as a symptom of the activity of the central nervous system. These authors believe that they have shown that a low blood-pressure is not an essential accompaniment of the clinical picture of shock. They, however, advise the methods of Crile for treating shock, with the exception of strychnia, which they believe is indicated before, during and after the operation involving shock. It seems evident from a careful survey of their work that their strong opposition to the findings of Crile is not well founded as they admit that there *were* some changes in the brain cells and that a degree of exhaustion of the nervous centers from which they could not recover is a possibility. They, therefore, admit two factors which, however, they were unable to prove experimentally.

A summary of the evidence in regard to the causative factors in the complex condition of shock may be stated somewhat as follows:

Shock is a condition of lowered vital resistance produced by varying factors, such as fear, injury or infection, of varying degrees. It is not possible to determine in a given case what physiologic function is first disturbed or what the exact sequence of pathologic changes are, but it does involve in its progress disturbance of the respiratory centre followed by disturbance of the oxygen-carbon dioxid equilibrium, lowering of body heat and depression of vasomotor tone resulting in a final paralysis or exhaustion of vasomotor activity, failure of respiration, cardiac failure and death.

Its treatment is based on a recognition of *all* of these factors. No one factor gives adequate explanation of the phenomenon.

THE CONTROL OF OPHTHALMIA NEONATORUM IN MASSACHUSETTS WITH SUGGESTIONS FOR THE IMPROVEMENT OF EXISTING CONDITIONS.*

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THIS paper is primarily concerned with the laws for the control of ophthalmia neonatorum in Massachusetts, the work which is being done in connection with these laws and with certain suggestions whereby, it seems to me, these laws may be still further improved. The necessity of such control is naturally more apparent to the oculist who is continually seeing babies incurably blind from this disease than it is to the other members of the profession who see these cases only occasionally or not at all. For an oculist to read a paper of this nature before a society composed principally of men in general practise, might under certain circumstances be regarded as a so-called "delicate situation." I believe,

* Read at a meeting of The New England Ophthalmological Society, Nov. 12, 1912, and at the semi-annual meeting of the Essex North District Medical Society, Jan. 1, 1913.