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Physiology and early treatment of burns

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The Physiology and Early Treatment of Burns

Burns have always been considered as one of the most severe accidents that the Physican has had to attend to. They are exceedingly painful and result in death or complications that are serious or horribly disfiguring. In the past, the doctors have been at loss to tell what caused the sudden death two, three or four days after the onset and when the patient was to all indications progressing satisfactorily. There would be a second severe shock, similar to the first, but rapidly becoming worse and in most extensive cases producing death with the same clinical observations as an acute toxemia. As early as 1823, physicans have been studying burns and their results but with no amazing results until up to the time of the world war. From then on the work has progressed with great rapidity and much has been proven. However there is still much left that is not understood and is explained by theory in a very unsatisfactory manner.

It might be well to classify types of burns briefly before the physiology and treatment is taken up. According to Dupuytren there are six groups listed according to severity. The first is where there is only a hyperemia or erythemia, the second group is where there has been the production of vesicles, third are those producing partial destruction of the true skin, fourth where there

is total destruction of true skin, fifth is where there is charring of muscles and lastly, where there is charring of the bony structures of the body.

It is with the more severe type of burn that this article deals, not because there are not the characteristic signs in the minor burns, but because the symptoms are so much more severe in the worse type of burn, easier to study and the results of treatment are more outstanding. Of course it is not only how deep the lesion is, but also to the extent that it covers the surface of the body and it's location on the body. It was this that led the earlier medical men to accept the theory that the cause of death in the severe cases was due to the destruction of the respiratory function of the skin and that alone. This is today known to be false, although it is agreed that the loss of respiratory function is a complication that plays some role in the production of death.

In the following discussion it is necessary to divide the clinical course of the injury into a number of stages as they are produced by different reactions of the body tissue to separate chemical and physiological entities. For this purpose I will accept the classification as given by W. C. Wilson in his article in the British Medical Journal, 2,91 of 1928. In this he gives four

stages.

I Shock (Initial or primary Shock)

II Acute Toxemia (Secondary or toxic Shock)

III Septic Toxemia

IV Healing

There are, of course, wide variations in the duration and importance of these stages depending on the extent and severity of the injury, and, more especially at the present time on the form of treatment adopted and at what stage in the course it was used.

SHOCK: This is the first stage in the clinical course of burns. This lasts from thirty-six to fourty-eight hours, and if marked, may be associated with a lucid mind and freedom from pain. It is more intense in children, in burns of the abdomen, trunk or the genitalis, and in burns involving a large area of the body surface. The main clinical symptoms are prostration, subnormal temperature, pallor, low blood pressure with a small rapid pulse and cold skin. It comes on immediately after the injury and in the majority of cases is slight in degree and passes off rapidly, to be followed by a period in which the patient appears to be well, even in some cases of extensive and deep burns. In eighty cases that terminated fatally in the Edinburg Hospital for Sick

Children, W.C. Wilson says, "We estimated that only two and one-half per cent of the deaths occured in this first stage and therefore obviously not a serious factor in the death rate from burns." Here the shock is produced entirely from the nervous involvement probably in the numerous nerve endings in the skin that has been injured. The vasomotor mechanism is upset and the condition produced resembles fainting or syncope, although it lasts for a greater length of time. Thus the patient that has a severe first stage shock is liable to have the same effect from any severe injury or emotional disturbance. The most severe initial shock that can be mentioned is that following injury to the testical or some other organ which is specially endowed with an abundance of sensory nerves. Now to what is this series of symptoms due? The earlier investigators were attracted to the pronounced fall in the blood pressure as the most outstanding symptom in shock, and attention was directed to its cause. This is caused by two things usually; peripheral dilitation of vessels or a diminished output of blood from the left ventricle. Crile, in his work on this matter, came to the conclusion that it was due to the former as the cause and that it developed because of a universal dilitation of the arteriols brought about by exhaustion of the tone of the vasomotor center. He concludes that the cen-

ter that "produces the normal alternation of consciousness and sleep is the mechanism whose alteration causes shock and exhaustion; and that in accomplishing restoration from exhaustion, this factor plays its part whatever the cause of exhaustion----whether emotion, exertion, physical injury, or infection, etc." He upholds the electro-chemical theory or that the nerve endings receive the impulses from an injury and transmits them to the brain in proportion to the depth and length of the injuring stimulus. When such occurs the brain cells produce acid by-products and by altering the acid base equilibrium upset the normal function of the brain itself. This alters the control of the various organs of the body, especially the vasomotor center and allows the arteriols to dilate. He upholds his arguments well by repeating the experiments when the tissue is injected with an anesthetic so that nerve impulses cannot pass to get to the brain. Since then however it has been shown by W.T.Porter, Morrison and Hooker, and Seelig and Joseph that such is not the case. They have demonstrated clearly that the tone of the vaso-motor center is practically normal in shock and that the arteriols are maintained, not in a dilated state but in a constricted state. This indicates clearly then that the fall of blood pressure is due to the decreased output of blood from the left ventricle.

They give very good proof for their statements. The rest of the symptoms of shock, such as the cold skin, pallor, etc, are due to the loss of the function of the brain to produce the necessary impulses to drive the various glands and muscles that perform the necessary physical and chemical activities by means of which the organism meets the vicissitudes of life.

This first stage then may be considered to be of nervous origin alone and there is no element of a toxemia entering into it as there is in the later stages which we will take up next.

The next stage or that of acute toxemia, is without doubt the most important and the most serious of the four stages. It was named by Robertson and Boyd as the period of toxic shock and has long been recognized as the real danger period. It has no definite time of onset after the initial lesion and its time of onset has been estimated to range from six hours to as great as sixty hours. W.C.Wilson in his article estimates the time as between twelve and twenty-four hours with, in the severe cases, an onset as quick as in six hours. This stage is responsible for about eighty percent of the deaths from burns and about seventy-eight percent of such cases died within fourty-eight hours after the infliction of the burn. In one case that Wilson cites, death occured as

early as fourteen hours after injury, although no shock was observed at the onset.

The clinical picture of an acute toxemia is quite typical and once seen should never be missed. The tem - perature rises, often very quickly and remains at that high level. 106° F. in children is not uncommon and in some cases has been known to rise as high as 109.8° F. followed closely by death. The patient is restless, the expression is anxious, the pupils dilated and the color dusky or livid. Vomiting is a common feature and the vomitus is often of a dark, coffee-ground ap- pearance. The blood pressure is low, the pulse becomes small and fast and the respiration rate increases. This course often increases rapidly and delirium sets in, followed closely by death. There are also some very im- portant laboratory findings. There is a diminished blood-volumn, concentration of blood cells in the per- ipheral blood, reduced blood alkalie and a concentration of the blood cells in their plasma medium, which is re- duced in volume. The clinical study of severe injuries by burns and trauma during the war convinced the obser- vers that the effect was not due to a nervous origin, since it did not occur immediately after the injury and that it was not of an infectious origin, because it was

so often well advanced before there was any evidence at all of an infection. Much evidence can be given to prove that it is of toxic origin. For example in the case of a severe injury to an extremity where there is great loss of blood and extensive mutilation a tourniquet is applied. As long as that constricting band is in place so that the blood can not circulate then there is no evidence of a toxic shock. But let the band be taken off and soon after the patient developes a typical toxic shock. But if the extremity is amputated before the band is released there is then no evidence of a toxemia. In other words, toxic shock developed if there was free circulation to the injured area, allowing the absorbsion of the toxins contained there. The most severe toxic shock was noted where there was envolvement of the muscular layers. The reason for this will be explained later in this article. The animal experiments of Bayliss and Cannon were very suggestive and enlightening. They crushed muscle and produced a condition simulating secondary shock and also noted that there was some substance liberated that had the power to lower the blood pressure. This did not occur when the vessels supplying the crushed area were tied off so that the circulating blood could not reach the body. However they found no substance in the urine that would explain it and since the effect was not permanent

they concluded that the body rapidly destroyed it or changed it to another form that is not harmful.

It has long been recognized that there are a number of substances that, when injected into the blood stream will cause a fall in blood pressure and a condition that is the same as the secondary shock noted in burns. Of ~~of~~ all these, the substance histamine shows the most ability in simulating the secondary shock. However there is no histamine as such in the animal tissues, but there is a closely allied substance and that is histidine. This can be easily broken down however, with the loss of one molecule of CO_2, into histamine. In other words it is a split protein. There is no formation of anti-bodies in the blood stream.

Avdakoff in 1876, found that a transfusion of blood from a burned animal caused the death of a healthy animal and thus first demonstrated that the blood contained a toxic substance. Later on by other experimenters, the arteries of two dogs have been anastomosed together in such a way that there is an interchange of their bloods. One animal was then burned and the effects were the same on both animals. The healthy animal went into secondary shock in the same manner as the burned animal. Further proof was given to this toxic theory when it was

found that rapid and radical excision of the burned area with removal of the toxic blood of the animal and transfusion of normal blood prevented the formation of shock and prevented the death of the animal.

As yet, the nature of the toxin produced is not known for sure, but it is well agreed that it is a product of protein lysis, due to the action of heat. There is abundant clinical, experimental and pathological evidence to prove this and some have already been taken up earlier in this article. As yet it has not been proved conclusively that this toxin is histamine or any other special protein compound but most observers are prone to believe that the substance is histomine. This, because it is so easily produced by the action of heat on histidine and because histidine is so prominate in all the body tissues. It is the greatest in muscular tissue and according to Crile this tissue is the one that, when burned, produces the greatest amount of secondary shock.

Even though the true nature of the offending toxin is not known, it has been proven that the secondary shock is due to a toxin produced in the site of the injury and when steps are taken to prevent the absorbsion of that toxin in the blood, then the patient does not go into the secondary shock that so often proves to be

fatal. With this in mind the treatment that is necessary is one that will prevent this absorbsion of harmful materials; one that is easily and quickly applied and will not too greatly mutilate the subject. It has been shown earlier that prompt and radical surgery of the parts involved is exceedingly helpful and to a great extent stops secondary effects. The use of a tourniquet will accomplish the same thing. But neither of these two methods are suited for the treatment of humans because they are too mutillating in effect. Then if it is not satisfactory to remove this effected tissue perhaps there is some chemical that when applied to the affected area will so change it that it is impossible for the harmful toxins to be absorbed. Such chemicals have been found in the form of protein coagulants. Such is tannic acid, picric acid, etc.

The third stage of burns is that of a septic toxemia. It may or may not be present, depending entirely upon whether or not there is an active infection present in the site of the wound. The severity of this stage, when it is present, then depends entirely upon the virulence of the organism and the extent to which the infection has spread. In many cases there is a severe bacterial infection which is very hard to combat satisfactorly. In the report by W.C.Wilson he states that of all

the cases he has studied, that fifteen percent of the deaths occured in ths stage. The symptoms of this stage are those of ordinary bacteriogenic toxemia.

The treatment of burns is one that has developed greatly in the last few years. This is due to the fact that the causes of the different stages have been more or less vague and treatment of a disease is based on the cause. Treatment used to consist of cleaning the wound and removing the dead tissue, then covering the effected area with an oil or paraffin and doing little else to the site of the injury. Of course the symptoms were treated as they appeared and often were sufficient enough to prevent the death of the patient. Blood transfusion was one of the means of combating the symptom of toxemia when it developed. Too often it was merely a means of prolonging the individual's life for a few hours, until the body absorbed more toxins from the burn again, so that the symptoms once more appeared. Because no one knew how to prevent this toxemia from starting and because severe burns would always carry this symptom, surgeons always regarded them with much anxiety and doubt as to their outcome. The introduction of tannic acid treatment in 1925 by Davidson of Michigan was a very definite advance in surgical therapy. The result was a quick drop in the mortality of these cases. Because of the

effectiveness of this treatment, I will spend the rest of this article on the treatment of burns by the tannic acid method.....

Treatment

In all cases of burns involving a large area of the skin the patient should be taken to a hospital for treatment. The immediate treatment, of course, is for the primary shock. It may be unwise for the patient to be removed from the scene of the accident for a few hours if the shock is very severe. In such a case the patient should be wrapped in a warm blanket, fluids forced by mouth and hypodermics of morphine given to stop the pain and quiet the patient. More often however the patient can be safely removed to the hospital and should be done with a minimum of care to the burn. Anything that can be done to the injury at this time is only likely to increase the shock.

Such dressings as carron oil and linseed should never be used when the injury is first attended. They do little good except to keep out the air and must be removed before any further treatment can be used. This means further trauma to the skin and an increase in the degree of shock. However it does diminish the pain for it keeps the cold air away from the area, and it is the cold air that increases the pain in such cases.

When the patient is in the hospital, he should be

placed in a warm room and all clothing removed. If morphine has not been given, it should be given right away, external heat applied and fluids forced, using hypodermoclysis and proctoclysis if the patient can not take fluids by mouth. In all cases of burns that are severe, prophylactic antitoxin against tetnus should be used. The initial dose should be from 1000 to 1500 milo. and it is wise to repeat within the week. The next treatment is often very painful and it may be necessary to give the patient a general anesthesia. It is unwise to give ether if shock is present and severe. For such cases an anesthetic of gas and oxygen should be used. The burned area should be thoroughly cleaned togetner with the surrounding tissue. All epithelium which is loose or blistered is removed. Special attention should be given to the edges of the lesion where loosened devitilized tissue covers what appears to be an area affected by a first degree lesion. This gets rid of tissue that is destroyed and will slough off in time and prevent absorbsion of toxins from that tissue. It also decreases the possibility of a secondary infection. However care should be used in this, for as Blair, Brown and Hamm remark, "Soon after the burn it may be impossible to differentiate between partial and full destruction of the skin, and this is one

reason against deep bebridement. Where any of the epitheal elements have been left, there will be a spontaneous healing with a very servicable skin;---- where there has been a loss of the whole thickness of the skin or derma, spontaneous healing occurs by an extension of the epithelium from the sides,-------- any lifeless tissue left will only decrease this growth." The raw surface that is now left is gently cleansed with some mild antiseptic. The most convenient for this purpose is ether. This not only dries the tissue but also will remove any oil that has been put on the injury before the patient came into the doctor's care. In no case should there be any vigorous scrubbing or rubbing of the raw area, because a profuse exudation of serum follows, which may interfere with the rapid formation of a coagulated layer.

Now it is the time to apply the tannic acid treatment. An aqueous solution of 2.5% tannic acid is used. This should be freshly prepared or at least not more than a week or two old, for it has a tendency to break down into gallic acid which nas no power to precipitate or coagulate proteins. It should be warmed before applying because as explained before cold substances have the tendency to increase the pain of a burn. The solution should not be much over five percent because strong

solutions have a corrosive action. There is no advantage of a five percent or stronger solution over the two and five-tenths percent solution. The warm solution is sprayed over the affected area with an ordinary mouth or nose atomizer. C.C. Robinson suggests that this be done once every twenty minutes until the tissue assumes a tan brown color and coagulation takes place. This occurs in about eighteen to twenty-four hours. H.T. Sutton has a little different method that he uses. First the burned area is covered over with flat sterile gauze pads. These are then soaked in a solution of two and five-tenths percent tannic acid by pouring the acid on them while they are in place. A small section of the gauze is removed at the twelfth, eighteenth and twenty-fourth hour. As soon as the red inflammed appearance of the area has subsided and the parts have assumed a light brown color all dressings are removed. He claims that this protects the wound from bacterial invasion until it has become partly coagulated and therefore less likely to develop a severe secondary infection. After the dressings are removed, the wound is sprayed every half hour until it assumes a dark brown color. However, no matter what the technique that is employed by these different men, they all agree that the tannic acid should be used until the skin is a mahogany brown. All during

this treatment the patient has the affected area exposed to the air but not in the open. He is best treated when he is under a 'cradle.' This is a frame that covers the patient and keeps the open air away from him and prevents the contact of clothes with the wounded area. Over this cradle there is spread a clean, sterile sheet, and on the under side of the frame there are electric lights. These keep the patient warm and help combat any shock that might develop, besides allowing the patient freedom of motion, lessened pain and easily accessable. It also drys up the exudations from the wound rapidly. In burns of the face, however, the eyes, nostrils and external auditory meatus are protected with moist wool during the spraying. Coagulation of the cornea is an accident to be guarded against. Artificial drying in this region can be dispensed with and also in general when the temperature is above 101°F. Sometimes the whole of the exposed area cannot be exposed to the air at once, as for example when the back and front are burned. In such cases the most severly burned area is the one that is exposed and the patient lies on the other that is less affected. The exposed area is treated by the spray method and the other by the method of C.C. Robinson or

the compress method. When these compresses are removed great care should be used so that the coagulum is not also pulled off. To prevent this it is best to soak each piece of gauze in the tannic acid solution for five or ten minutes before trying to remove them. From the time that the coagulum has formed completely there is little need for any special local treatment. The coagulum must not be damaged or loosened for this would defeat the purpose that it has; so in children it is a good thing to tie their hands and feet to the bed or cage and keep all bed clothes from rubbing against the affected area. Any new blisters that develop should be opened and treated as the rest of the area has been. Care should also be taken that urine and fecies do not soil the injured area as this is very apt to release the toxins in the coagulum and start a septic process. If the burn is in the area of the buttocks, a pillow should be put under the hips to support them and the bowels can be regulated by rectal lavage.

If the symptoms of secondary toxic shock should develop in the patient after the injured area has been treated, there are other procedures that can be carried out and they prove to be very helpful and life saving. Of course from the beginning, as mentioned before, there

should be the forcing of fluids into the body by any method that is best employed at the time. In severe cases four thousand to six thousand c.c. of water should be given to an adult patient every twenty-four hours for three or four days. Often the oral route is satisfactory alone. Morphine should never be given in this stage, for it would only be depressing an already severly depressed body and it might prove fatal. Diaphoretic drugs are also contraindicated as they tend to further concentrate the blood, which is already concentrated by the extravisation of fluids from the blood vessels. The out-put of urine will be seen to be diminished and blood chemistry will show a nitrogen-retention when the toxemia starts. This is a signal for the doctor to pour more fluids into the blood stream, both for the dilution of the toxins and to bring the blood volumn up to the normal. Two means are used. The first that I will mention is the one that is the least used but one that has shown very good results in the hands of those that have used it. This is the introduction of one thousand c.c. of a normal glucose solution or normal salt solution intervenously. Blair, Brown and Hamm use the glucose method of "withdrawing some toxins from the blood and diluting the rest by the continuous venoclysis of five percent glucose. H.T. Sutton says about this

method," The intervenous medication has much to recommend it. Its effect is immediate------It is not advisable when giving fluids intervenously, to give it faster than twenty-five c.c. a minute. If sugar is being given it should be even slower, six to twelve c.c. per minute. The temperature of the solution should be 105°F. It will be lowered two percent from the container to the vein. The value of intervenous saline lies not only in its diluting and diuretic effects but in the addition of NaCl to the blood------Glucose solution has certain advantages over the salines. It is not only diluting and diuretic, but it is also nourishing. It is an efficient way of alleviating acidosis and serves to relieve toxemia." All fluid introduction tends to restore the blood concentration to its normal levels. It will eliminate toxins, and dilute them. With the increase of blood fluids there is an increase in the vasculartone of the vessels due to this dilution of the inspissated viscid blood.

The use of blood transfusions in most cases of traumatic shock is par excellence. The method used by the majority of authors is the exsanguination transfusion of Robertson and Boyd. About three hundred to five hundred c.c. of blood are withdrawn from the body and about 1000 cc. of fresh blood are injected. In a child the only way

to bleed them is to pass a needle or cannula through the saphenous into the femoral at the fossa ovalis. If this site is destroyed then one must use the jugular vein. The cubital vein is not surficient. This early transfusion and exsanguination transfusion may react not only by diluting a circulating toxin but also by binding it or destroying it to a certain extent. In that way it is better than the intervenous glucose.

The most difficult problem in the treatment of burns is sepsis. The incident of sepsis by the cleaning of the burn vigorously with the use of strong antiseptic solutions is unchanged. W.C. Wilson in the survey of all of his cases makes the statement that there are as a rule no signs of sepsis in burns of the second degree, either locally or generally, nor in many of the third type have any such symptoms. In a considerable proportion of deeper burns however, there is evidence of sepsis. Most of these are local infection signs and are manifested by some reddening of the edges of the burn and the seepage from the edges of the coagulum of a little sero-purulant discharge. If, as is usual, the general condition remains satisfactory, the coagulum may be safely left alone, and no special treatment is necessary...Gross infections rarely occur except in association with the formation of slough s.

in deep burns. If a pool of pus should collect beneath the coagulum, it will cause it to be lifted up at the base. Then it is easy to strip it off or cut a gash in the coagulum to allow the escape of the fluid. At times it will be stuck down so that this is not easily done and the thing to do in such a case is to cut it away with scissors. Formerly it was recommended to soften the coagulum with vasoline first but this procedure has led to the release of some of the toxins and unfavorable signs and symptoms arise. Therefore it is no longer recommended by the doctors. W.C. Wilson states that under no conditions should a moist or wet dressing be applied to this coagulum when there is presence of a sepsis. He says that the injury must be kept dry at all times, all blisters should be opened at once, no fluid should be allowed to remain under the exudate, no moist dressings or even vasoline should be applied over the affected area. "The real explanation of this association has not yet been ascertained.....Davidson observed the same signs after the application of boric fomations over the coagulum.....It seems reasonable to infer that the toxin previously imprisoned in the coagulum becomes soluable in the presence of moisture and gains access to the blood stream."

In direct opposition to this treatment of Wilson is the article by Blair, Brown and Hamm who say, "If there is much infection and crusting, the patient may be put on a Bradford frame, which is elevated to allow the irrigating fluid to run off over the end of the rubber sheet into a bucket at the foot of the bed. Every hour or so, from five hundred to one thousand cc. of saline solution is poured over the patient....Surgical solution of chlorinated soda, acriflavine hydrochloride, hexylresorcinol, or any other desired antiseptic may be applied on loose gauze dressings. Any adherent gauze is left in place to be soaked loose in the next bath." They even recommend hypertonic salt baths daily where the whole body is immersed for thirty minutes. H.T. Sutton is against the use of any antiseptics when there is an infection not for the reason that it will release toxins, but because "it will lead to hard and contracting scars." He advocates the use of Dakon's solution instead to clean up the tissues and dissolve the necrotic tissue. He also recommends the use of sunshine as it has the power to dry up secretions and abolishes the stench that often occurs in suppurating wounds.

It also seems in the opinion of most men that the use of braces and traction to keep the extremities, if

they are involved, from being drawn up when the scar tissue contracts, is useless. It is for later treatment to do this and which will not be discussed in this article.

Summary

In conclusion of this article, we find that the most important time of treatment in most of the cases of burns is duringthe third stage. As explained in thepages before, this is the time whenmost of the deaths occur and they are caused not from the loss of skin area, but due to the presence of a strong toxin in the blood stream. Therefore it is quite evident that the line of treatment to be carried out is one that will either destroy these toxins, or prevent them from entering the bloodstream in the first place. Due to the war experience, a very effective plan of treatment has been put in to effect. This is a combination of two lines of attackt. First to coagulate the tissues and prevent absorbsion, and second to dilute the toxins already present in the circulation. To do this, tannic acid seems the best as a coagulant. Care is then taken that nothing is done that might release these bound up toxins, or in other words soften the coagulum. The forcing of fluids and transfusions or injections of saline or glucose complete the treatment

by diluting and combating the toxemia.

Since this line of treatment has been used, the mortality has dropped greatly. Burns no longer are feared as they used to be by the medical profession .

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