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Trauma and The Living Cell

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Oration on Trauma

TRAUMA AND THE LIVING CELL are the "leit motif" of his lifework, says Joseph Trueta, Oxford, Nuffield professor of orthopedic surgery, University of Oxford. From his student days at the University of Barcelona Faculty of Medicine and surgical apprenticeship under Professor Manuel Corachan, Professor Trueta acquired a life-long interest in the behavior of the tissues and in the cells which characterize these tissues. When early in his career he became surgeon in charge of an organization dealing with accidents, where on an average of 40,000 patients a year were treated, Professor Trueta observed the ways in which the injured cell reacts to trauma and the means whereby the surgeon can alleviate or add to the "suffering of the cell." This experience and data collected during the Spanish Civil War, World War II and in the laboratories and clinics at Oxford during more than 20 years are synthesized in the accompanying Oration on Trauma made October 13, 1960 at the Clinical Congress in San Francisco.

APART FROM THE DAMAGE OF DIRECT INJURY which may kill the cells outright, or so disorganize the tissue that it cannot survive for long, most of the cells which decide the final outcome in trauma are only slightly affected at first. No reference, of course, is made here to the massive tissue death following severe damage to main arteries, for these ischemic changes which cause tissue necrosis are not the object of our attention now. We are thinking instead of the larger group of posttraumatic conditions in which the tissue cells have been affected either directly by, or immediately following, a single, severe trauma.

Professor Trueta

Let us consider the following example: A car accident has caused a compound fracture of the leg. From the first moment a number of epithelial cells have died at and near the edges of the skin wound. A variable amount of subcutaneous and intramuscular connective tissue and fascia has been disrupted, vessels divided and muscle bundles cut and stripped away from their bone or tendinous attachments. The tibia and fibula are comminuted and some fragments are deprived of the vascular connections. Added to these liabilities are the bacteria which have penetrated into the disorganized focus of fracture.

Everything has happened in one rapid moment, and all moves quickly from then on. The ruptured vessels bleed into the focus, only the excess of blood which cannot be retained among the tissues draining spontaneously away. The unavoidable posttraumatic changes occur, or would occur if other factors were not to intervene. The ruptured ends of the majority of local vessels would close by the clotting of the blood and the contraction of the vessel wall. Shock and hemorrhagic anemia decrease the blood pressure, which helps to stop bleeding. The so-called posttraumatic inflammatory reaction mainly attributed to the change of local hydrogen ion concentration, the proteins extravasated from the blood, the production of histamine and to some extent undefined sympathetic stimulation all enter into play. But it appears certain that at the bottom of this, it is the amount of tissue damage and cell death which causes the inflammatory reaction.

Tissue fluids accumulate both by means of an increased transudation and decreased lymph flow.

We had the privilege many years ago of helping to demonstrate, following the work of Drinker,[1] the mechanism by which lymph accumulation occurs in the limbs. With J. M. Barnes[2] we found that by canalizing the large lymphatics of the rabbit in the groin, the flow of lymph would cease while the limb was kept totally immobile, whereas the flow was resumed as soon as movement began, or even when isotonic muscle contractions were elicited by faradic stimulation. We know that the pain on movement after trauma is responsible for the muscle quietness, the main cause of fluid retention.

In the absence of infection, the wound would enter into the second phase of repair and could be considered for all practical purposes not very different from a closed fracture. In such cases the inflammatory changes, including pain and increased transudation, are, as mentioned, mainly provoked by cell disorganization and death caused by the accident either directly or by the local disruption of blood supply, even when only minute vascular branches are damaged.

But here we must consider two new factors which either alone or acting together may hamper the reparative efforts and, on too many occasions, are the real cause of disaster. The first of these is the eventual penetration of bacteria into the wound and, the second, wrong surgical management of the injury and the patient. Of the two factors the more difficult to avoid is, or should be, the penetration of bacteria and dirt to the midst of the disorganized tissues.

It must be remembered that bacteria are endowed with a desire for survival which compares favorably with our own. They recognize the quality of the food offered to them and readily begin reproduction as soon as conditions of the medium permit. But, of course, a living tissue in physiological conditions, bathed by fluids rich in antibodies and in phagocytic cells, would have the upper hand provided that, first, the number of bacteria were not too massive or too aggressive from the beginning and, secondly, that local conditions did not depart too much from the normal.

Numerous dead cells and tissue fibers, retained blood and, it must not be forgotten, stagnant lymph enhance the unfavorable local conditions, and in these indirect ways bacteria succeed in increasing the death rate or the degeneration of cells. We know well how infective arteriolar thrombosis and excessive compressing edema, by cutting from their local blood supply other areas of healthy tissue, are the principal factors responsible for the invasive nature of the infection in badly handled open fractures.

Although experience compels the surgeon to readjust many of the convictions of earlier days, I still firmly believe as I did over 30 years ago when I began to handle trauma, that the equation on which success or failure depends is based on the relationship of the three main factors involved—trauma, vessel, cell. Of these three elements trauma is present from the beginning and cannot be controlled, apart from attempting to prevent it

[1]Drinker, C. K. and Field, M. E. Lymphatics, Lymph and Tissue Fluid (Baltimore: Williams & Wilkins Co., 1933).

[2]Barnes, J. M. and Trueta, J. Absorption of Bacteria, Toxins and Snake Venoms from the Tissues. Lancet I, May 17, 1941: 623.

Fig. 1—Early organization of woven bone with a perfused vessel is demonstrated. Endothelial cells are laying down bone on all visible sides.

Fig. 2—Osteoblasts which originated from vascular endothelium form lamellar bone on all visible sides.

from occurring. But the second element, the vascular damage, while partly inseparable from trauma, may be indeed minimized by proper surgical care. This is essential if we attempt to preserve alive all the cells which have escaped the original injury.

We are now coming to the contribution of the surgeon which may hamper or accelerate the reparative capacity which lies dormant in all living tissue. We refer to the surgeon's ability, based on knowledge, to prevent further damage to the cell from being added to that unavoidably caused by the initial injury.

Let us examine in some detail the basic alterations brought about by the trauma which may be favorably influenced by the proper handling of the case. For this analysis we will proceed from the surface to the depth of the traumatized area in the hypothetical open fracture referred to previously.

Skin: The main characteristic of the tissue which covers and isolates the body is its resistance to damage by external injury; this is attributable to its thick corneal surface, elasticity and its rich blood supply. Thus, an injury must be very severe to cause more than limited damage to the skin, apart from that of the zone near the cut edges of a wound. Besides, the epithelial cells of the skin have the property of surviving for relatively long periods despite the transitory suppression of their normal sources of nutrition. This is seen every day with the use of the split skin graft to cover larger areas of the body. Provided the receiving area is rich in vascular tissue not isolated from its surface by excessive fibrous proliferation, in the absence of infection the epithelial cell preserves its vitality nourished by the transudates of the surface of the wound.

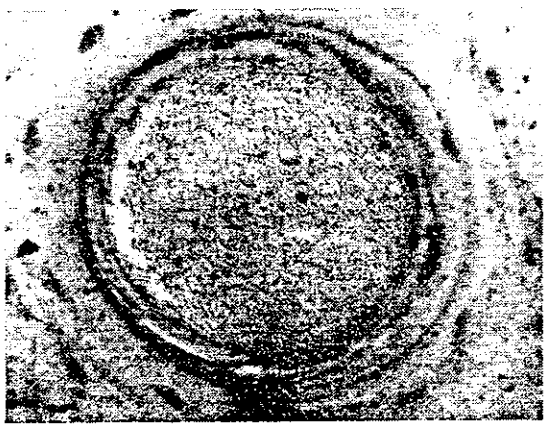

Fig. 3—Transverse section of perfused vessel shows its endothelial cells in process of laying down bone in all the periphery of the vessel.

Another consideration of great importance for the survival of the epithelial cell is firm contact between the bed and the implant.

It is commonly supposed that the contribution of gentle pressure to the success of grafting is due to the avoidance of fluid interposition between the implant and bed. This is surely true, but another and probably more important reason which is solely related to the quality of the wound surface exists, and explains the benefit of pressure. I refer to the superior quality of the vascular-connective tissue covering a clean wound even after only two days from the time the wound is inflicted, if appropriate pressure has been exerted on its surface. This pressure, evenly distributed, prevents the formation of exuberant granulations and assures the development of a flat bed of vascular tissue on which epithelial cells will readily survive. The exudation decreases and with this the superficial connective cells do not cause fibroblastic metaplasia, thus the endothelial vascular wall remains nearer the new epithelium which either progresses from the wound edges or is supplied by the skin graft. We have evidence of the benficial effect of pressure upon wound healing in the treatment of varicose ulcers of the leg. Reduction of transudation by elevation of the limb is also helpful in these cases.

Muscle: The behavior of the muscle fiber after trauma is in marked contrast with that of the epithelial cell. Some apparently slight disorganization, even a simple reduction of the blood flow through the muscle, may cause permanent damage which goes from fibrous degeneration to total muscle necrosis. Nevertheless, the capacity of

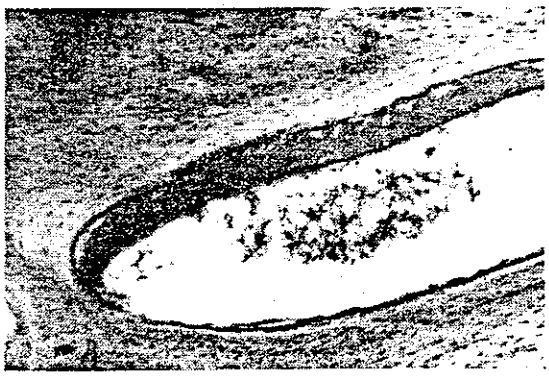

Fig. 4—Lamellar bone around a marrow space shows on its upper part a well perfused vessel laying down a new lamella.

muscle to regenerate is apparently much greater than it has for many years been assumed. In addition, we know that the muscle deprived of blood is an ideal culture medium for anerobic bacteria. Consequently, exposure of the depths of the wound is necessary to permit a thorough examination of the vitality of muscles and fascia in the treatment of wounds. Detailed, meticulous excision of all devitalized muscle fibers is essential to obtain a safe recovery but compression by fasciae may also contribute to muscle ischemia when the intermuscular spaces are engorged by tissue fluid. This is the chief danger of primary skin suture in these severe cases.

Bone: The enormous use in contemporary surgery of bone grafts and the clinical and experimental study of fractures have provided us with excellent means for investigation of the behavior of bone as a tissue, and of bone cells following the severe trauma responsible for fractures.

We know now beyond any doubt that the bone cells die if the bone they inhabit becomes detached from its natural soft tissue connections. This is due to their inability to withstand being deprived of blood even for only a few hours, probably less than eight. In any bone graft or totally detached bone fragment, which naturally has lost its blood supply following a fracture, practically all osteocytes are killed before new vessels invade and revitalize the bone by proliferation of the endothelial cells of the vessel wall. Only on some occasions a row or two of superficial osteocytes may be seen apparently alive. Thus, the incorporation of the dead bone

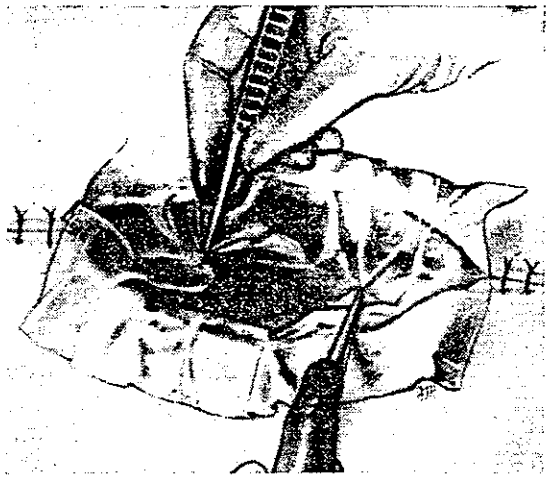

Fig. 9—A large piece of fine-mesh "calico" absorbent gauze is placed over wound at end of excision.

fragment or graft will depend on the rate and quality of vascular penetration which forms in both cases the same type of new tissue, namely, the provisional callus. How the vascular cell intervenes differently either in the neighborhood of the bone or of the bone marrow spaces is illustrated by Figures 1 through 8 (Pages 74-77).

In open fractures infection is—with wrong treatment—the main cause of severe complications. Provided infection is avoided and local conditions do not offer obstacles beyond the normal capacity for repair, such as massive soft tissue interposition between fragments or a bone gap too large for the callus to bridge, final bone reconstruction will occur.

Bone repair is mainly a vascular enterprise; consequently an appropriate general and local blood

Fig. 5—Shown above is transverse section of a perfused vessel with an endothelial cell shortly after its division.

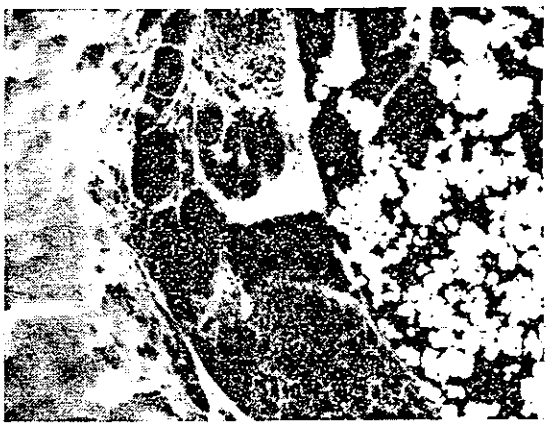

Fig. 6—Vessel perfused with fine barium suspension, micropaque, is on right and two endothelial cells are adjacent to bone in calcified matrix.

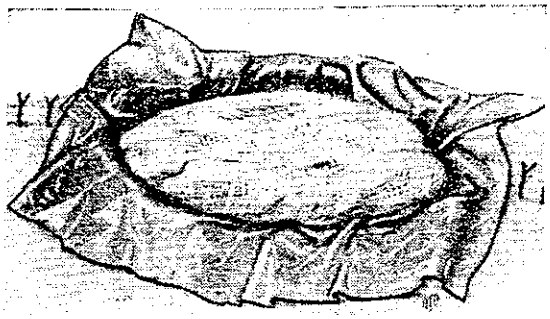

Fig. 10—Ordinary absorbent gauze or cotton wool presses "calico" firmly against all irregularities of wound. Drainage by suction is thus established to prevent even small fluid retention on which bacteria may thrive.

flow must be encouraged from the beginning. Anemia, persistent shock and low protein intake all work against supplying good blood in appropriate amount to the cells which most need it.

Lack of correct immobilization causes repeated rupture of the proliferative vascular ends and exhausts the reproductive capacity of their endothelial cells; fibrosis with lack of union is frequently the final outcome of a fight between the vessels and the surgeon, where the vessel is defeated.

Infection does not produce in the bone the same dramatic adverse results as it does in the muscle. But by preventing the vascular invasion, infection is the main cause of irreparable osteocyte death and bone necrosis; thus a persistently unhealthy wound is usually the expression of devitalized bone.

Summarizing the chief directives for winning the battle fought at the cellular level for recovery after trauma, the following technical points are put forward, all aiming at the protection of the life of the cell in severe open injuries.

- Prevent and treat shock which by decreasing the circulation of the damaged tissues leaves them at the mercy of bacteria and reduces further the number of surviving cells.
- Clean the skin and wound with soap and water to eliminate bacteria and foreign bodies.
- Exercise extreme conservatism with the skin, removing only its torn edges, but extirpate dead muscle; and exercise reasonable conservatism in handling bone.
- Only exceptionally close by primary suture of the skin an accidental wound causing severe muscle damage. In good hands this may be done in peace time injuries; but never in war injuries.
- When the wound is initially left open, to achieve the best drainage of its depths use calico absorbent gauze which maintains the surfaces of the tissues dry (Figures 9 and 10).
- Use firm but gentle external pressure as it is essential to the good health of the vascular-connective tissue and the epithelial cells of the wound surfaces.
- Immobilize and protect wounds when necessary with plaster casts or splints, and elastic bandages. These insure healthy vessels and a well drained wound for the first days until it is covered by skin suture or by grafting within one week from infliction of wound.
- Systematically elevate the extremity, in the

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Fig. 7—Vessel with red corpuscles is on right, with two cells of vessel wall at middle adjacent to osteoblast in process of self burial to become an osteocyte.

Fig. 8—End of calcification around an osteocyte in lamellar bone is demonstrated. Notable are numerous canals through which cytoplasmic processes establish contact with those of nearby cells.

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absence of major vessel injury. This reduces edema, contributes to the normality of cell metabolism and to the satisfactory end result.

The foregoing are the principles and practice of a method of wound treatment which I promulgated during the Spanish Civil War and later recommended and used during the second World War.

This method has little in common with that recommended by Dr. H. Winnett Orr for the treatment of established osteomyelitis, with which it was unfortunately confused. Cleaning of the wound, excision of devitalized tissues, and drainage with dry absorbent gauze were meant to prevent infection while Orr designed treatment to control suppurating bone sepsis. When this latter treatment was applied to freshly inflicted wounds and fractures it caused frequent, severe complications and always an offensive smell.

The principles underlying the correct management of damage to the living cell induced by a single violent episode have been considered up to this point.

The surgeon is also forced to face damage to the living cell caused by repeated minor trauma. Let us mention but one example taken from the field of orthopedics—osteoarthritis. This condition is the result, primarily, of improper distribution of pressures over the joint cartilage.

As the cartilage has no vessels, nourishment of the chondrocytes is effected by the inhibition and diffusion of fluids from the surface of the cartilage —synovial fluid—and from the depths—transudates from the bone. When the distribution of pressures in a joint is so disturbed that a particular segment of the articular cartilage is subjected to excessive and persistent pressure, then the balance of the nutritional mechanism is disrupted and cell death ensues. Finally, cartilage erosion and vascular invasion from underneath take place, and the deformity and sufferings of osteoarthritis become established.

It would be out of place here to enlarge on these facts for they have been repeatedly described and discussed, but they are brought up to show that surgeons, and particularly the orthopedic surgeons, are faced all the time with biological problems situated at cellular levels. For instance, when performing an osteotomy in the treatment of osteoarthritis of the hip, we are changing the distribution of pressures into the hip joint, and of the blood supply to the head of the femur, helping the survival or regeneration of the damaged cartilage cells, principally responsible for the disintegration of the joint.

Our increasing understanding of these problems is not only now saving more surgical patients than ever before, but is also raising surgery to the category of an applied science. There is thus full justification for training the young surgeon as much in the fundamental biology of the tissues he handles as in giving to his hands the same control and skill required of the musician.

This association of fundamental knowledge and manual dexterity constitutes not only the attributes of a surgeon of today but the greatest hope for the surgery of the days to come.