Use of Ceramics in Surgical Implants

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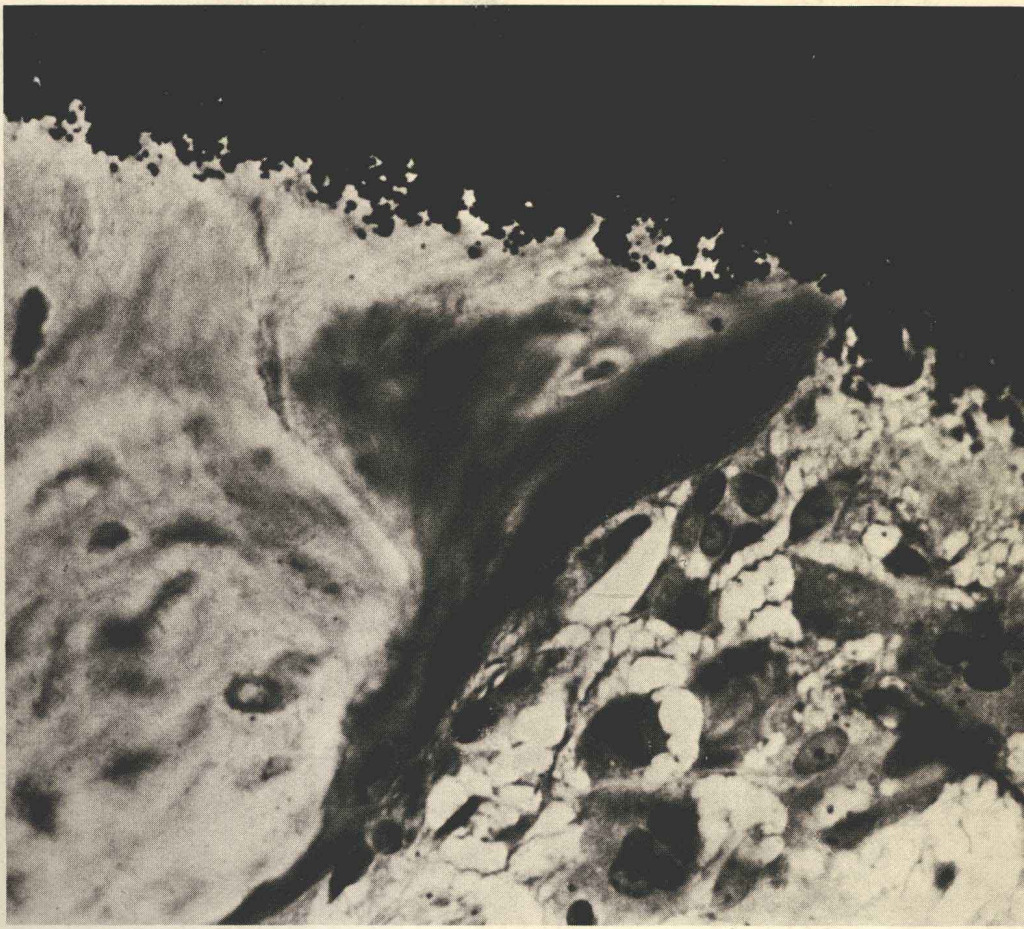

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PREFACE

The First Clemson Biomaterials Symposium entitled, "Use of Ceramics in Surgical Implants" was held on January 31 and February 1, 1969 on the Clemson University campus.

The meeting is recognized as being the first meeting of the series which is now referred to as the "Annual International Biomaterials Symposium" and out of this meeting has evolved the Society for Biomaterials.

The purpose of the symposium was to stimulate interest in the area of "Bioceramics" by acquainting materials engineers with materials problems that exist in surgery and by reporting scientific work which has demonstrated the feasibility of employing ceramics as materials of construction for surgical implants.

Several classic papers were presented at the symposium which are often referenced in the literature. The symposium served as a major stimulus to the development of bioceramics. There are a number of investigators who contributed papers at the first symposium who at the time were new to the field of biomaterials who are now considered to be among the leaders of the biomaterials research community.

Because the symposium on the Use of Ceramics in Surgical Implants has played such an important role in the evolution of new materials of construction for artificial organs, it is felt that the proceedings should be formally published.

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CHAPTER 1

BIOMATERIALS THE CASE FOR CERAMICS

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Introduction To Biomaterial

Throughout time one of the greatest puzzles facing mankind has been man himself, that is to say, the human body and how it functions. Technological and scientific advances have unlocked many of the mysteries of the human body and have shown it not to be a magical unit but an intricate array of physical and chemical processes. Our ever increasing understanding of the body has naturally associated with it a desire to repair the human body damaged by diseases, accidents, old age, and birth defects. Such repair is envisaged as diverse as synthetic bones[1] and as extensive in scope as the implantation of artificial hearts.[2]

One of the major problems facing workers in the design of prosthetic devices is the choice of materials of construction. The requirements for any type of permanent implant are in the general sense the same; the implant must be resistant to attack by physiological fluids (dilute saline solutions that are often quite acidic in the region of a flesh injury); the material must be strong enough to withstand the forces imposed on it during its life expectancy; the material must be capable of being formed or otherwise shaped into the variety of necessary configurations,[3] the material should cause neither allergic nor toxic reactions; the material should not alter the electrolytic composition of plasma and tissues; the material should not interfere with the body's normal defense mechanisms nor trigger the development of cancer; and finally, the most limiting criterion of all, the material

should not promote blood trauma, blood clotting nor denaturing of plasma proteins.[4]

When prosthetic materials are placed in the body, two points of view may be considered. One is the effect of the physiological environment upon the prosthetic material, and the other is the effect of the prosthetic material and its corrosion or degradation products upon the fluids and tissues of the surrounding environment.

Metals have in the past been the predominant material used as internal prostheses. With the advent of polymer science and the development of more and varied polymers, it was recognized that the resilience which many of these materials afforded could be put to much better use in the replacement of soft tissue than could the metals. Thus, within the past twenty years both metals and plastics have been used extensively in the human body—metals predominantly for their strength properties in fracture fixation and plastics for their resilience in soft tissue repair.

As knowledge has been gained about the complex activities which take place within the human body, the demand for new and improved materials which can better withstand this highly corrosive environment has greatly increased. This demand, plus their generally excellent chemical stability, has, within only the past few years, forced the long overlooked ceramic materials into areas of biomaterials research. The use of ceramic materials for prosthetic application in the human body is presently in the experimental and limited clinical stages. Their obvious chemical and physical properties would initially indicate ceramic materials for surgical implants to be best suited as bone and tooth prostheses, and it is those applications which have shown the most promise in investigations to date.

Medical Polymers

A large variety of organic materials have been investigated as materials of construction for prostheses, including acrylic plastics, polystyrene, polyethylene, polyurethanes, methyl cellulose, polyvinyl alcohols, nylon, Teflon, Dacron, Orlon, Mylar, Silastic, Bakelite, ivory, beef bone, stag horn and kangaroo tendon.

Medical polymers are presently the most successful material for soft-tissue implantation in the cardiovascular, respiratory, digestive, genitourinary and nervous systems. Medical polymers are pliable, light in weight, thermal insulators, easily moldable, readily procurable, and inexpensive. One of the most successful uses of surgical implants is in the replacement of mural and septal defects of the heart with Teflon and Dacron patch materials. Despite the many desirable properties of present day medical polymers, the degradation of medical polymers by the physiological environment and the lack of compatibility between medical polymers and the

physiological environments are major problems in the development of artificial organs.

Effect of environment on polymer implants.—Although Atlas and Mark[5] state that polymer implants are insoluble and are not absorbed by the body, but rather the toxic materials are actually antioxidants, stabilizers, and the low molecular weight aromatic additions, Oppenheimer et al[6] detected carbon-14 in the urine of rats after intramuscular implantation of tagged polystyrene, polyethylene, and poly methyl methacrylate. The carbon-14 had to have been freed from the main chain of the polystyrene and polyethylene and from the ester side chain of the methacrylate, emphatically demonstrating the metabolism of polymers by the body. Certain polymers such as the polyurethanes, superpolyamides, polyacylonitriles, and polyvinyl alcohols have been found unsatisfactory for use as soft tissue replacement because of deterioration and fragmentation.[7] Chemical analyses of various polymer implants which have shown deterioration indicates cleavage of the polymer chain. Both oxidative degeneration and hydrolysis have been described as probable mechanisms of polymer degradation in implants.

Harrison[8] found that over a three-year period nylon fabric grafts in portions of the thoracic aorta of dogs lost most of their original strength, and similar grafts of Dacron, Orlon, and Teflon lost measurable amounts of their original strength over two year periods. Nylon was also reported by Maloney[9] to lose a large percentage of its strength when used as suture material in the abdominal wall of humans. Further, Szilagyi[10] described decreases in tensile strength values of woven tubes of both nylon and Dacron implanted in aortas of dogs for up to 54 months. Mirkovitch[11] et al implanted polyurethane intramuscularly in dogs. The materials showed an extreme loss in tensile strength after eight months and after sixteen months, the sample had disintegrated to the point where tensile strength could not be measured. The rapid deterioration of polyurethane was quite unexpected due to the polymer's high resistance outside of the body to such corrosives as ozone, oils, radiation, strong acids, and alkalis. Leininger[12, 13] and coworkers implanted films of five plastics intramuscularly in the flank of dogs for 6, 11, and 17 month periods. After seventeen months of implantation, polyethylene decreased in tensile strength from an original value of 2,700 psi to 1,930 psi, while over the same time period, nylon showed an even greater decrease (9,300 psi to 5,200 psi).

The mechanical properties of Mylar and Silastic, after seventeen months of implantation, did not appear to be significantly effected by the physiological environmtnt. After 17 months, Teflon showed an increase in tensile strength from an original value of 2,950 psi to 3,720 psi and a decrease in percent elongation at break from 320% to 250%. This stiffen-

ing effect is attributed to the ingrowth of fibrous tissue and has also been found to occur in polyurethane and polyvinyl (Ivalon) sponges used for mammary augmentation. In these cases the tissue ingrowth caused shrinkage and subsequent hardening or the sponges. In other applications under stress, the stiffening may often result in fragmentation of the implant.[12,14]

Organic materials in general lack sufficient strength for extensive use as structural prosthetics. In their limited application as skeletal prostheses, polymers have so far been found to creep under relatively small loads and fail to hold fractured bone fragments in their proper orientation. Struthers[15] implanted polyvinyl (Ivalon) sponge in various skeletal tissue of young adult dogs. In these studies it was found that although fibrous tissue and growth of new bone infiltrated the surface spaces in the sponge, the sponge became compressed and displaced.[15]

Effect of plastic implants and degradation products on surroundings.— The decomposition or degradation products of polymer implants in many cases have been found to be directly or indirectly associated with various abnormal tissue reactions. Both systemic and local polymer administration have been demonstrated to be related to tissue irritation by some type of chemical or physical mechanism.

According to Woodward,[16] among the experimentally demonstrated potential dangers of systemic polymer administration are: (1) antigenic phenomena, (2) reticuloendothelial hyperplasia, hepatosplenomegaly, anemia, and ascites, (3) hypertension and nephritis, and (4) tumor formation at sites of disseminated polymer depositions.

Polyvinyl alcohols injected subcutaneously in rats were found by Hall and Hall[17] to produce anemia, hepatosplenomegaly, hypertension, nephritis, and ascites. The production of these disorders was thought to be mainly due to low solubility of the polymers in the body fluids. These same investigators found that glomerulonephritis and hypertension could be induced in rats by injection of methyl cellulose.[18] Through several means of administration of dextran and polyvinylpyrolidone, Heuper[19] stimulated the development of carcinoma-appearing disorders in various organs and organ systems.

Among the primarily local untoward results of polymer implants are tumor induction and carcinogenicity,[20] excessive inflammation, decomposition of the polymer to yield locally toxic products, and sluggish non-physiological or non-optimal repair of tissue damage.[16,20,21] Lack of biological reactivity does not appear to preclude untoward responses to implants, such as local tumor induction in rodents.[16] Numerous experiments involving implantation of various kinds, shapes, and sizes of polymers have been conducted using rats, mice, and hamsters, with rats being the most popular experimental animals.

After experimenting with Bakelite discs, Turner[22] was the first investi-

gator to report that local tumor induction often followed implantation of polymers in rats. Oppenheimer[23] et al observed development of sarcomata in and about kidneys of rats wrapped in cellophane films. By the subcutaneous implantation of cellophane films they further encountered production of malignant mesenchymal tumors (fibrous arcomas) in rats and connective tissue tumors in mice.[23,24] Bering and Handler[25] also reported the development of connective tissue tumors in hamsters after implantation of cellophane films at subcutaneous sites.

Johnson[14] reported on reactions to cellophane, Dacron, nylon, Orlon, polyethylene, and teflon in the human body. Although none of the tissues showed evidence of malignant changes, implants of these materials were reported to be associated with various degrees of inflammatory reaction and development of fibrosis. The inflammatory lesions were not characteristic of one plastic, but were common to all types. Cases of functional failure of arterial prostheses were accompanied by thrombosis, hemorrhage, and excessive fibrosis. In nine examples in humans of cellophane or polyethylene wrapping of the aorta extending over periods of two months to four years, postmortem examination showed significant local thrombosis present in six cases and hemorrhage in four cases. Fibrosis and infiltration of the vessel with chronic inflammatory cells were present in all instances.

The observation that no proven case of induced malignancy is known to have resulted in humans from permanent implantation of artificial materials is difficult to reconcile with the large number of reports[19–29] suggesting that any organic substance has the ability to increase the incidence of tumor formation in laboratory animals.

In addition to rapid deterioration and low abrasive resistance resulting in minute wear particles, the nylons have been found to be highly irritating and produce an excessive foreign body reaction.[16,30] Polyvinyl (Ivalon) sponge implants in humans have been associated with ulceration of overlying tissues and the presence of foreign body giant cells.[14,15] The disintegration of polyurethane arterial prosthesis has been found to be accompanied by formation of aneurysms.

The principal causes of functional failure of plastic vascular prosthetic devices in humans are thrombosis, hemorrhage, and excessive fibrosis. At times, the hemorrhage can be shown to be due to fragmentation of the plastic with loss of mechanical integrity.

Braley[30] points out that presently the only polymer materials which have proven to be suitable for long-time soft tissue replacement in the human body are certain high grade silicone rubbers. For reasons not yet determined these materials apparently do not cause foreign body reactions and are not metabolized by living organisms. The tensile strength of Silastic is less than 1,000 psi.

Most experimental implantations of polymers, however, are followed

by diverse inflammatory responses, and local or systemic histotoxicity from degradation products. Absence of immediate tissue reaction to implants in test animals does not signify acceptance of the polymer by the host tissue, as eventual tumor development at the implant site may emphatically demonstrate.

Physical and chemical properties, purity, resistance to degradation, physical form, and site of application, among other factors, of polymers all affect the tissue responses and compatibility with physiological fluids. According to Guccione[4] Teflon, Dacron and Silastic all cause more or less pronounced clotting. There is no evidence that prosthetic materials in the heart or blood stream exert any direct effect on white cells or plasma.

The development of completely implantable artificial organs awaits the development of prosthetic materials which are more compatible with soft tissues and physiological fluids than those now employed in surgical implants.

Prosthetic Metals

In most fractures, traction, plastic casts, or other types of external fixation are sufficient to immobilize the fragments and to allow for early healing with a minimal amount of stiffness of the joints. However, there are many cases in which the extent of the damage precludes the use of simple external fixation, and some type of internal prosthetic device is both necessary and desirable in order to obtain proper healing. In injuries where severe bone damage has resulted, the need for some type of permanent implant material is indicated which may take up the functional role of the missing or severely damaged natural structure.

Many and diverse structural materials have been placed in the human body in attempts to aid the body in the self-repair of skeletal disorders. By far the predominantly used materials for bone prosthesis have been, and are still metals. All types of the more common metals and alloys have been tried in prosthetic application at one time or another. Gold, silver, copper, lead, zinc, cadmium, tin, iron, nickel, aluminum, magnesium, vanadium, vanadium steel, bronze, brass, steel plated with other metals, Ticonium (an alloy of nickel, cobalt, chromium, 6 percent molybdenum), Elgiloy (a complex cobalt base alloy), Vitallium (a cobalt base alloy containing chromium and molybdenum), titanium, zirconium and types 302, 304, 316 and 317 stainless steels have been employed at various times in various shapes and forms in hopes of aiding bone healing.[3] The metals have taken the form of nails, screws, nuts and bolts, staples, bone plates, intramedullary pegs, wires, bands, but other configurations.

The highly corrosive and immunologically sensitive type of environment into which an internal bone prosthetic material is placed and in

which it is expected to coexist, and to behave as a functioning integral member, discounts most of those metals previously listed from potential use in permanent implant applications. Corrosion or similar chemical degradation caused by the action of body fluids and tissues on the implant not only changes the physical properties of the implant, but, in return, the products so formed are toxic, causing allergies and/or carcinogenic responses and subsequent isolation and rejection of the foreign body.

Aside from toxicity due to degradation products other sources of irritation include physical and mechanical incompatibility of the implant with the bone and tissue. For example, differences in the coefficient of friction between cartilage and an implant in a joint can easily initiate an area of irritation. Likewise, differences in flexibility can cause irritation and destruction of bone in areas where contact is made with an implant material.

Design is a very important area of consideration in the fabrication of implants. Sharp corners, edges, and crevices must be avoided since these areas act as centers of stress concentration and localized corrosion, or other types of chemical degradation, particularly when subjected to large alternating and repeated stresses in the highly corrosive body fluids.

Another important consideration is the surface condition of the implant material. Differences in strain, crystalline structure, and the presence of surface impurities may all lead to differences in solution tendencies. The more homogeneous the surface the more resistant it will be to disintegration. If the implant materials were to be subjected to a homogeneous environment, the design and fabrication of a piece with continuously uniform surface properties would not be as difficult. However, it is not a static homogeneous environment, but a very dynamic nonhomogeneous one. As described by Wickstrom,[31] it is an "angry" environment encouraging exchange of electrolytes and "salty" conducive to corrosion. It is oxygenated with differences in oxygen tension between different portions. Oxygen is brought to the tissues, and ions in chloride solutions swirl past a given area with considerable speed. It is well known that the composition and pH of the body fluids changes from time to time. There is a pH change occurring in damaged tissue, and a potential difference between injured and normal tissue has been recorded. Murry and Swenson found that following necrosis and tissue injury there is a pH fall to between 5.3 and 5.6.[32] A gradual rise ensues with development of pH close to 7.35 in approximately ten days. From this it is apparent that since internal prosthetic appliances are used only in injured and necrotic areas and additional tissue damage is unavoidable in the process of implantation, all internal prostheses are subjected to a rapidly flowing, highly corrosive environment of changing pH.[32]

Most metals are not static, durable materials. Their natural tendency is to revert to the oxide. This reversion in the presence of H_2O is called corrosion. Metals placed inside of the body are aided in their reversion tendencies by the body's internal environment; and that environment, as emphasized previously, particularly in areas of injury, is one of large and variable stresses and dynamic, highly corrosive fluids and tissues. It has been found to be unique in its rapid degradation of many materials previously thought to be unaffected by highly corrosive fluids.

Most metals are either not resistant to corrosion by body fluids (e.g. aluminum), react with body tissue (e.g. copper) or lack sufficient strength to serve as a structural prostheses (e.g. gold).[3] As an example of what is meant by reaction with body tissue, consider nickel. Nickel causes inflammation and discoloration of tissue, retards reparative growth, produces excessive scars and erosion of bone. Due principally to their greater corrosion resistance while maintaining strength values comparable to other metals which have been used as internal prostheses in the past, types 316 and 317 stainless steels and cobalt-based chromium-molybdenum alloys (predominantly Vitallium) are, with few exceptions, the only metals presently used in artificial implant applications. One exception is the limited use of titanium or titanium alloys in heart valves and orthopedic appliances. Titanium has been reported to show an affinity for newly formed bone tissue in experimental cases.[33]

Effect of environment on metal implants.—Stainless steels, as is the case with many other metals, depend on the presence of a closely adherent oxide surface layer for their resistance to corrosion. When continuous, this thin layer of corrosion products protects the metal from any further oxidation, in what might otherwise be a highly corrosive environment. Due to the mechanism of this type of protection the corrosion resistance of the metal and the ability to maintain the integrity of the oxide film depends on the continuous presence of oxygen in the environment. Thus, the stainless steels are passive in corrosive salt solutions such as the body fluids as long as the oxygen tension remains relatively high and is uniform over the entire surface. However, even the most corrosion resistant stainless steels—type 316 and 317—are susceptible to differential oxygenation, a situation which does occur from one point to another on the surface of implants in the body tissues. Whenever two pieces of a multi-component metal implant come into contact there exists a stagnant area depleted of dissolved oxygen. Such areas are most commonly located between the screws and plate of fixation devices, whereas the remaining surface area of the plate is exposed to a high oxygen concentration continuously flowing through the adjacent fluid and tissues.[32,34,35]

When differential oxygenation is present over the surface of stainless

steel several means are available for corrosion to occur. Where the surface is in contact with a low oxygen environment the equilibrium of the reaction of metal with environmental oxygen is shifted in favor of the breakdown of the protective oxide film. The resulting exposed metal acts as an anode to the surrounding protected areas which are cathodic. Pitting corrosion commonly takes place on the anodic unprotected metal and is continuously accelerated by the presence of chloride salts. If the low oxygen potential is due to metal to metal contact, pitting and crevice corrosion may also result from damages to the surface oxide film which may have occurred during the process of implantation, or from abrasion and relative motion while in place. In such areas the absence of sufficient oxygen prevents the damaged surface oxide layer from renewing itself.[32,34,36,37]

Pitting corrosion is a form of intensive local attack in which cavities with approximately 1:1 depth to diameter ratios develop in the metal surface. It is affected by differential oxygenation and increased chloride content. Uhlig[38] experimentally demonstrated that above pH 2.8, type 316 stainless steel in a 4 percent sodium chloride solution receives the deepest pits at pH values between 6 and 7. These conditions are comparable to those of the chloride solutions found in the body.

Crevice corrosion is another form of localized attack occurring in shielded areas, usually between two surfaces. Surface damage caused by contact between the two metal pieces ruptures the protective film leaving the crevice in an active condition shielded from oxygen.

Colangelo and Greene[39] microscopically examined the corrosion of type 316 stainless steel orthopedic implants removed from humans as a matter of normal surgical practice. Of 53 devices 24 (45 percent) were found to have corroded and 4 of these had fractured. Of 23 multi-component devices examined 21 (91 percent) exhibited corrosion, indicating that the probability of escaping corrosion in a multi-component device is rather small. The predominant form of corrosion was crevice attack which occurred in 42 percent of all possible sites. Three of the fractured devices were definitely identified as having fractured through fatigue failure. Physical irregularities which were identified as manufacturing defects were shown to act as focal points for subsequent corrosion.

Fink and Smatko[40] tested screws and plates of 302 and 316 stainless steel and Vitallium in various corrosive media. They subjected these materials to physiological saline, serum, serum saturated with excess sulfanilamide, serum inoculated with *Staphylococcus. aureus* and serum acidified with potassium acid phosphate to pH 5.0 to 5.5. No noticeable changes in the weight of the metal pieces were apparent until they were subjected to stress, after which time perceptible weight losses were recorded. The term stress corrosion is applied to such cases of corrosion in which there would

be no significant corrosion damage in the absence of stress. Due to highly localized attack in the area of stress, it is possible for alternating stress to crack an implant in a corrosive environment well within the safe stress ranges determined under noncorrosive conditions. Stress corrosion cracking is the most common form of actual failure encountered in stainless steel implants. It occurs intergranularly where there is subsurface torsional and tensile stresses, predominantly in areas which have been notched, stamped, or otherwise subjected to stress in design and manufacture, such as in the threads and notches of screws.[34]

Whenever different metals are linked together in an electrolytic medium galvanic corrosion results. This type of corrosion within the body is less apt to occur today than it was in the past before galvanic activity was understood, or even realized. However, even at the present time, due to mistaken labeling and sometimes ignorance of the phenomena, mixing of metal does occasionally take place in internal fixation devices. Scales et al,[44] during corrosion studies of Smith-Petersen nails removed from patients in a London hospital, found that of 65 nails studied 11 were labeled incorrectly as to type of steel. A classic example is the use of Vitallium (cobalt-base alloy) in combination with stainless steel (iron-base alloy). The corrosion due to metal transfer is also caused by the galvanic effect. In this case bits of one metal have been transferred to the metal implant which is of different composition. The transfer usually occurs during handling or during the implantation, where tools used to drill holes, twist screws into place, or hammer pins into position are made of a different alloy from that of the implants. Even different batches of the same type of alloy, however, may vary enough in composition to cause a potential difference sufficient to initiate corrosion and affect local nerve impulses.

Still other variables may set up potential differences on the surface of a single metal when subjected to the body's electrolytes. Variations in the hardness of crystalline structure from one point to another and the presence of precipitates and impurities on the surface act as preferential sites for corrosion activity. A scratch or other type of damage on the metal surface acts as an anode to the surface metal around it. Also, different concentration of metal ions may be set up because of varying conditions in the nonhomogeneous electrolytic environment. Any difference in the concentration of ions in contract with different parts of the same metal surface may induce corrosion currents. When stress is also brought to bear upon the implant, all of these areas may be conducive to metal failure.

Many years of experience have shown that metals placed in the body to aid in skeletal support can often fatigue and break. Continuous remodeling of bone tissue along the line of increased stress gives natural bone a resilience and resistance to fatigue which is unobtainable in the artificial

implants. The repeated stresses to which living bone can adjust are enough to initiate intergranular corrosion which leads to eventual fatigue of the metal implant in contact with the bone.[31]

None of the presently known metals in the usual implant designs are capable of standing up to stresses applied to them in recurrent load bearing across an ununited fracture. In clinical cases where excessive loading has been present, metal rods, pins, and fixation plates have been found to undergo fatigue failure within a few weeks to a few months after insertion. Even the minor flexing occurring normally in bones can cause enough repeated stress in an adjacent metal implant to result in its failure. Intramedullary rods, used to hold long bone fractures during healing, have often been shown by x-ray to be broken in two after several years of repeated small bendings within the healed bone.[32,42] Alloys used for orthopedic implants are subject to fatigue and have fatigue limits generally, less than half their ultimate tensile strength. They also show fatigue notch sensitivity so that an implant device with a geometric notch may fail by fatigue with maximum loads as low as one-tenth the static breaking load of the device.[36]

Fatigue failure of metal fixation devices within the body is due to the presence of several factors. The difference in flexibility between bone and metal under the application of cyclic stresses while in the presence of a highly corrosive environment initiates the slip and intergranular corrosion which in a short time produces failure.

Effect of metal implants and corrosion products on surroundings.— Macrocorrosion is common with stainless steels[35,41,43] and microcorrosion is seen with all types of metal alloys including Vitallium.[44] This latter type consists of the deposition of metallic elements in the tissue around implants. The existence of high concentrations of constituent metallic ions has been demonstrated in adjacent tissues for all metals which have been and are currently[1] used as artificial implants in the human body. Laing and his associates have done considerable work in this area. In spectrochemical analysis of tissues adjacent to type 316 and 317 stainless steel implants, they found large concentrations of iron which were not present in control samples.[45] In rabbit studies constituent elements could always be demonstrated in tissues adjacent to any metal implants. Microscopic analysis of these tissues showed definite zones of tissue reactions after 6 months in contact with metal, and the degree of reaction was proportional to the amounts of constituent elements released in corrosion.[46]

In numerous clinical cases, areas about metal implants, without warning develop sudden inflammation and pain, and the only relief is removal of the metal. This and other evidence indicates the probable existence of critical levels of metal ion concentrations, below which the tissues can