may be a central bone abscess, with slight or extensive central necrosis; or the whole shaft, for a variable extent, may undergo complete destruction. Should the patient survive, an abscess will develop in the soft tissue, which may rupture and continue to discharge pus and sequestra for years.

So soon as drainage is established, either naturally or by surgical interference, reparative processes begin, both in the endosteal and the periosteal tissue. The osteogenetic layer of periosteum rapidly proliferates and produces a new layer of bone, which may take the form of long splints or ensheathing tubes, corresponding to the extent of cortical destruction.

If the peptonizing action of bacterial and inflammatory products were capable of rapidly dissolving or breaking up and separating the dead bone, as it does the slough in similar infections in the soft tissues, the rapid proliferation of the endosteal and periosteal tissues, with its subsequent transformation into osteoid and fully developed bone, would soon repair the injury inflicted and restore the bone to an almost normal condition. The presence of the sequestrum, separating these actively proliferating tissues, prevents their union at a time when repair could be made perfect. Even in total necrosis, when the sequestrum is removed early, the periosteum alone is able to produce an almost perfect anatomical shaft.

But, under ordinary circumstances, when nature has finally disposed of the dead product, or it has been tardily removed by the surgeon, the activity of the regenerating tissues is arrested by its more or less complete transformation into bone, and the resulting cavity in it is lined with a layer of granulation tissue, which possesses but little of its original regenerative activity; besides becoming the home of numerous pyogenic organisms, which, from time to time, acquire increased virulency and light up fresh inflammation, leading to further destruction of bone, or else by acting as an irritant lead to sclerosis of the newly formed involucrum.

Infective osteomyelitis, as a rule, begins with sudden local pain, throbbing in character and increasing gradually in severity. The situation is usually near the end of one of the long bones. The pain is increased on pressure or jarring the limb. Swelling of the soft parts soon follow. The surface becomes reddened and pits on pressure. The contiguous joint becomes swollen, contains increased fluid, though cultures secured by aspiration of the joint remain sterile. Later, in the course of the disease, suppurative arthritis may supervene. The constitutional disturbance is prompt and indicative of