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TRAUMATIC PANNUS

I. Macroscopical and Microscopical Changes after Experimental Reconstruction of the Joint Surface

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Pannus formation is a reply to changes and defects which have developed during various pathological processes in joints. The pannus is described as a fragile, cellular and well-vascularized tissue that grows from the synovial lining or from the opened vascular spaces of the subchondral bone. Later it loses its original cellularity and vascularisation and changes into a fibrous tissue, which can undergo further differentiation, depending on the functional load of the damaged joint. According to the tissue activity related to the articular surface, one distinguishes a chondrolytic, ossifying, ankylosing, fibrositing pannus. The development of the pannus can be caused by a series of different factors:

1. Traumatic lesion of the articular cartilage. If the defect of the cartilage is so deep that it perforates the subchondral plate, a vascular and cellular tissue grows into the defect from the subchondral vascular spaces (Carlson 1957, Landells 1957, Banks 1966, Akeson 1969, Campbell 1969).

2. Traumatic damage of intraarticular structures such as menisci and cruciate ligaments. In such a case the granulation tissue develops in the area of the lesion and overgrows the nearest articular cartilage (Helfet 1959).

3. Ischaemia affecting the joint. During such changes both kinds of pannus develop, the intraarticular and the medullary pannus. The first one is created by proliferation from the synovial membrane or from intraarticular structures and covers the surface of the joint cartilage. The second kind develops in the intramedullary spaces, penetrates

through the articular cartilage and later becomes connected with the intraarticular pannus (Rutishauser & Taillard 1966).

4. Immobilization of the joint. Irreversible damage of the cartilage results from prolonged therapeutical or experimental immobilization of joints. The cartilage becomes covered by a connective tissue growing out either from the capsule or from the medullary spaces after the perforation of the cartilage (Evans & Eggers 1960, Akeson 1961, Hall 1964).

5. Continuous extensive pressure on the joint surface destroys articular cartilage. In such a case the defect may be repaired by the deeply situated chondrocytes which have escaped death or by granulation tissue growing out from the subchondral marrow spaces (Trias 1961, Thompson & Basset 1970).

6. Articular cartilage is damaged not only from excessive pressure but also from absence of weight bearing. The relief of contact in joints results in early subchondral vessel invasion of the cartilage matrix and loss of the zone of calcified cartilage (Hall 1969).

7. Damage of the articular cartilage during degenerative processes. In such cases the pannus develops mostly from the synovial lining and covers both the healthy and necrotic cartilage in those areas that are in contact with the synovial membrane (Salter & McNeil 1965, Nako-něčnyj 1967, Batra, Charnley 1969, Mankin & Lippiello 1970).

8. Damage of a healthy articular surface by contact with the synovial lining. The cause can be a lesion of the capsule, a one-term or continuous overloading, or a maximal excursion of the joint. Cooper (1961) describes "Kissing or contact hip syndrome", which can manifest itself as a productive or destructive lesion of the anterior portion of the neck and margin of the head of the femur.

9. Rheumatoid arthritis. The inflammation of the synovial membrane is the cause of the pannus formation in rheumatoid joints. This pannus, which consists of an apron of vascular granulation tissue composed of proliferating fibroblasts, collagen fibers, numerous small blood vessels and variable numbers of inflammatory cells, overgrows the articular cartilage and replaces it (Hamerman 1969).

The aim of our experiments was a detailed investigation of the pannus and changes occurring in it after transplantation of an autograft or homograft of the articular non-weight-bearing surface.

MATERIAL AND METHODS

For our experiments 90 adult mongrel dogs were used of a mean weight of 12 kg. The whole anterior portion of the distal end of the femur, the osseous component of which was 6 to 8 mm thick, was transplanted. A homograft was placed into the right knee and into the left knee an autogenous graft taken from the other knee joint. Homografts were preserved for 7 to 14 days in paraffin oil at a temperature of 4° C. Homografts were treated in different ways before transplantation: by conservation only (Fiala et al. 1965), by drilling the graft through (Fiala & Bartoš 1967), by drilling a part of homogenous cancellous bone and its replacement by the autogenous one (Fiala & Herout 1965), by washing of the cancellous bone and its impregnation with autogenous marrow (Fiala & Bartoš 1971), by washing of the cancellous bone followed by impregnation of a fibroblastic substance (Bartoš & Fiala 1971) and by X-ray irradiation of the graft followed by washing and impregnation of the cancellous bone with autogenous bone marrow (Fiala & Herout 1972).

The grafts were fixed to the bed with a single wire loop. Before sacrificing, the blood vessels of each experimental animal were injected with a mixture of Indian ink and gelatine. The grafts were removed with their beds and fixed in a 4 per cent neutral solution of formalin. After decalcification in a mixture of formic and muriatic acids the preparations were imbedded into paraffin. Histological sections were stained with hematoxylin and eosin, Alcian blue and by Mallory's, van Gieson's and Goldner's techniques. The vascularization was studied in unstained sections 250 μ m thick.

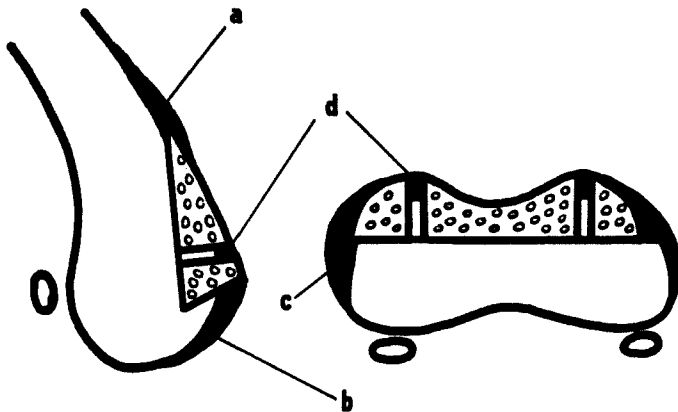


Figure 1. The distal end of the femur. The graft is spotted, the pannus marked in dark. Pannus formation: (a) at the connection between the cartilage of the graft and the periosteum of the bed; (b) at the junction of the cartilage of the graft with the bed; (c) at the connection of the graft basis and bed; (d) in the drilled defect.

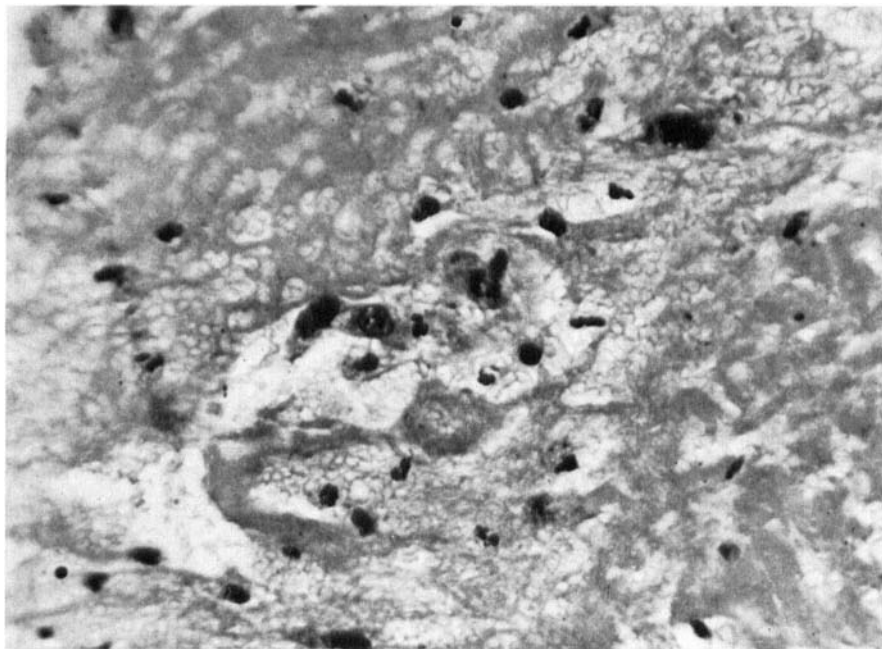


Figure 2. Pannus from the basis of the autograft 4 days after transplantation (1/67-A). The network of the fibrinous bundles among which numerous erythrocytes, their remnants and dark-coloured nuclei of degenerated leukocytes can be found. In the middle of the picture there is a mitosis of a fibroblastoid cell. Van Gieson, $\times 400$.

RESULTS

In the joints opened 2 to 5 days after operation, the pannus was found in all areas where the graft was in contact with the bed (Figure 1). The greater the difference between the size of the graft and the bed, the larger was the amount of the pannus. The pannus was of a fragile consistence and adhered only slightly to the articular cartilage. Its *bright red colour* distinctly reflected against the surrounding darker tissue, where, after injection of the blood vessels of the cancellous bone with Indian ink, the cartilage of the bed looked dark blue and the synovial membrane black. Only the cartilage of the graft preserved its pinkish colouring.

In the transverse thick sections blood vessels injected with Indian ink were found only in the bed and in the regions of junction of the graft. The blood vessels penetrated neither into the graft itself nor into the pannus.

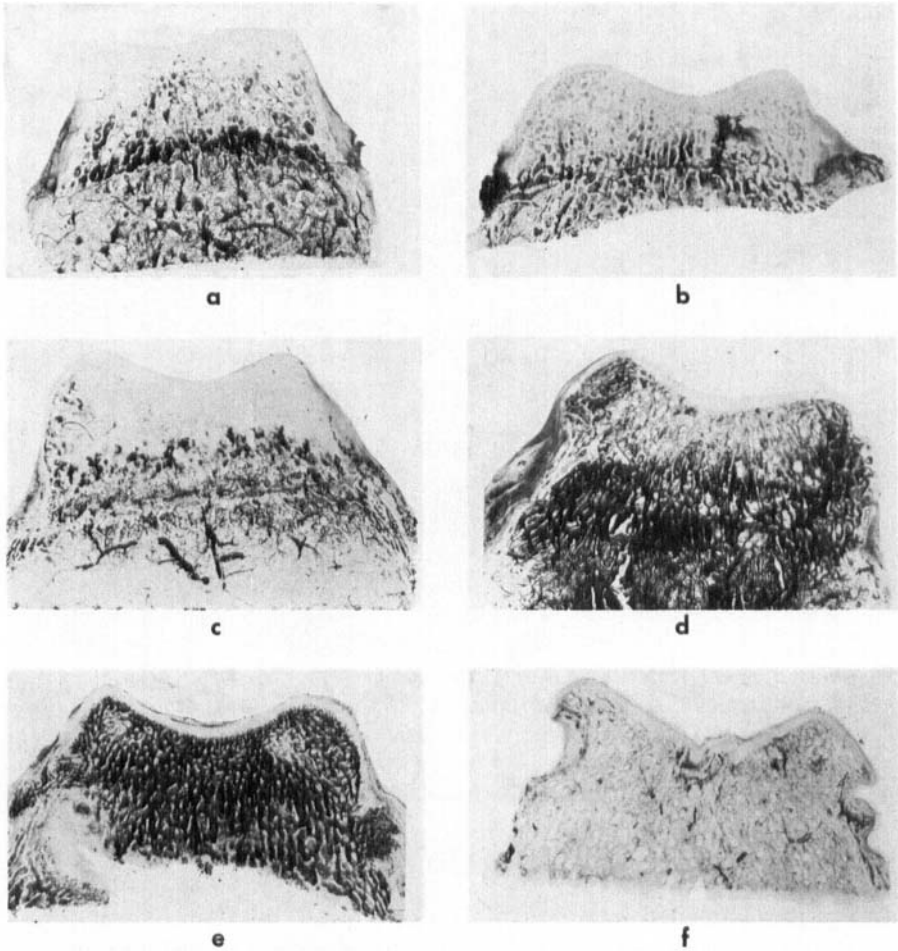


Figure 3. *Histotopograms of the junction of the graft with the bed. (a) Autograft 7 days after transplantation (10/65-A). The connection of the graft and the bed is characterized by high vascularity. Pannus on the right side of the graft is not yet vascularized; on the left side the initiated vascularity can be seen. (b) Autograft 9 days after transplantation (91829-A). On the right side the pannus fills in the incongruity between the graft and the bed. The initiated blood vessel invasion. (c) Homograft 14 days after transplantation (5/67-H). The lower half of the graft is vascularized and the blood vessels penetrate the pannus approximately to the same level. (d) Autograft 21 days after transplantation (7/65-A). The graft is vascularized nearly in its whole thickness and the dark colour of pannus shows the great amount of blood vessels in it. (e) Homograft 28 days after transplantation (91027-H). The plastic pannus on the right side of the junction of the bed and graft is formed by an island of the osteoid tissue without any orientation of its trabeculae. (f) Homograft 28 days after transplantation (98028-H). The lytic form of the pannus can be seen on both sides of the graft. Unstained sections 200–300 μm thick, blood vessels are filled with mixture of Indian ink and gelatine. $\times 2$.*

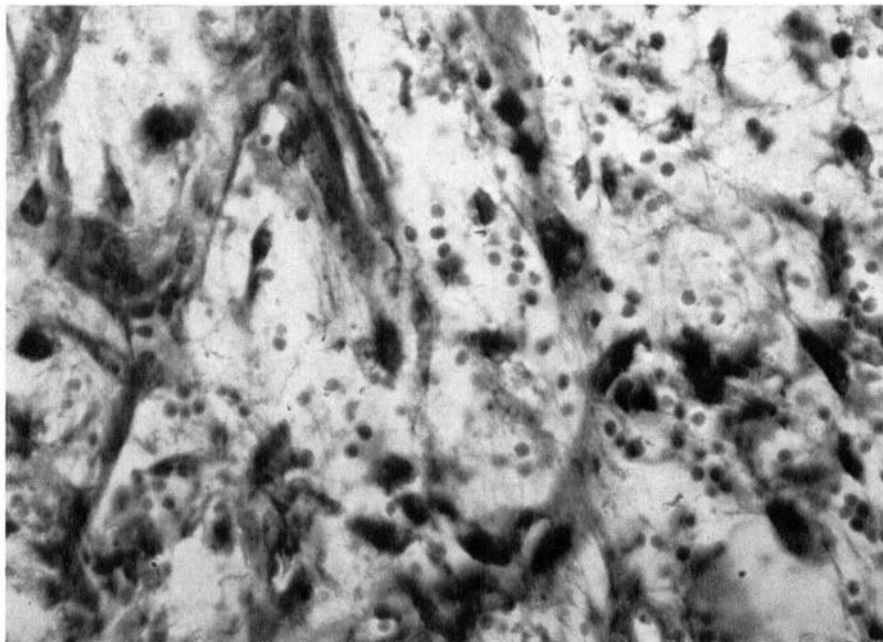


Figure 4. Pannus from the basis of the autograft 7 days after transplantation (2/67-A). Near the penetrating blood vessels there are many fibrocytes, some of which are found in mitoses. The fine collagenous fibres are without a distinct orientation. Van Gieson, $\times 400$.

The histological picture showed a network of fibrin bundles of various thicknesses with cellular elements between them. Most of these elements were erythrocytes, many of them dying off with only the cell membranes left over. Polymorphonuclear leukocytes, also partly degenerated, were marked only by pyknotic sphericles left over by their nuclei. Mononuclear cells were mostly of the histiocytic type. Some of the fibroblastoid cells were observed during their mitotic division (Figure 2).

From the 6th day after transplantation the marginal parts of the pannus adjacent to the periosteum were losing their original sharp bright-coloured demarcation (Figure 3 a, b).

In the histological pictures the original stroma of the pannus was preserved only in these areas that had not been penetrated by the blood vessels. The number of erythrocytes decreased remarkably. In the areas of blood vessel penetration numerous small empty spaces developed. Around the vessels a rich cellular population, mostly fibro-

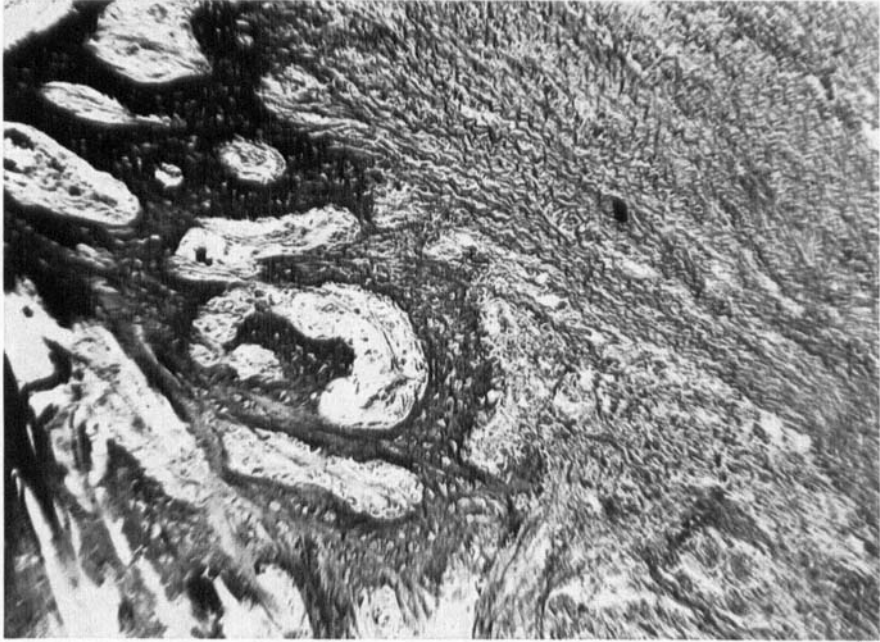


Figure 5. Pannus from the basis of the autograft 21 days after transplantation (4/67-A). On the picture is the junction of the cancellous bone of the graft with the pannus. The bundles of thick collagenous fibres running parallel with the surface pass into the trabeculae of the graft. Mallory, $\times 100$.

cytes, can be found. Many mitotic divisions and fine collagenous fibres without a distinct orientation were observed (Figure 4).

In the second and third week the unsharp demarcation of the pannus also began to manifest itself in the areas where it adhered to the articular cartilage of the graft (Figure 3 c, d).

In histological slides the pannus tissue started to acquire a pattern of a maturing granulation tissue. Among the cells, fibrocytes prevailed. The tissue that adhered to the bone contained bundles of collagenous fibres running in parallel with the surface of the bone. Nearer to the surface the collagenous network was finer and thinner. In some spots, mostly near the base of the graft, rudiments of thin osseous trabeculae and small marrow spaces were formed (Figure 5). Near the surface of the pannus, histiocytic and fibroblastic elements were found as well as single erythrocytes.

Within the time from the 4th to the 8th week after transplantation the consistency of the pannus changed. The originally brittle and soft

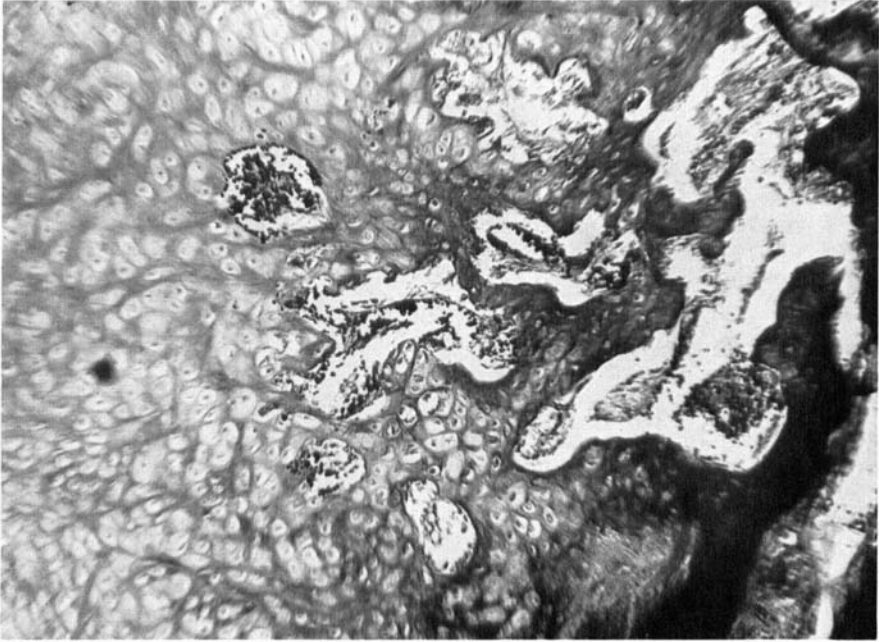


Figure 6. Pannus from the basis of the autograft 2 months after transplantation (7/67-A). An island of fibrocartilage at the base of the pannus is formed. Mallory, $\times 100$.

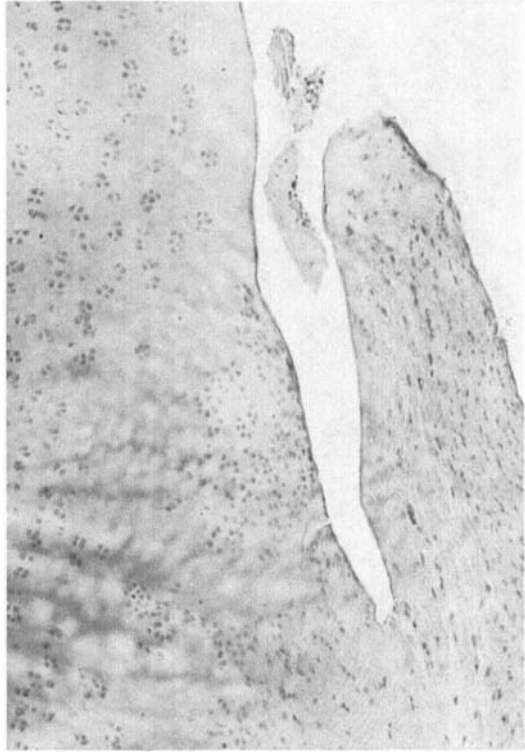
tissue gradually became more consistent, and it was no longer possible to separate it from the cartilage without damaging it.

At this time, the two main kinds of the pannus could already be distinguished: the plastic one and the lytic one. The plastic form developed most frequently in the areas of proximal connection of the graft and the bed. On places of the original pannus, deposits were formed protruding above the articular surface firmly adhering to the base and covered with a glistening white tissue. The lytic kind of pannus was mostly observed at the sides of the graft as a defect penetrating more into the graft than into the bed. This form of the pannus was frequently found in homografts (Figure 3 e, f).

In histological slides the pattern of the pannus was that of a mature scar tissue. It was formed by a network of dense bundles of collagenous fibres; fibroblasts and blood vessels were very rare. In numerous places fibrous cartilage (Figure 6) or young bony tissue developed.

In regions where the articular cartilage was overlaid by the pannus,

Figure 7. The side of the autograft 70 days after transplantation (85554-A). Cartilage at the basis is necrotic and partially replaced by the ingrowing pannus. Between the cartilage and the tongue of the fibrous pannus can be seen a gap which prevents the acrtilage from devastating action of the pannus. Hematoxylin and eosin, $\times 40$.



necrosis of the superficial layers and ingrowth of the pannus tissue into the necrotic cartilage were found (Figure 7).

In the junction of cartilage of the graft with the cartilage of the bed the avascular pannus was replaced by the granulation tissue growing from the opened marrow spaces of the subchondral bone. The cellular and vascular pannus filling the gap between the graft and the bed continued to replace the necrotic cartilage of margins of graft and the bed (Figure 8).

In the time between the 4th up to the 12th month the appearance of the pannus tissue practically did not change. Only in disintegrated grafts were protrusions of whitish tissue found, covering the defects imperfectly and only in part. Histological pictures showed large islands of fibrous tissue, fibrous cartilage and mature bone tissue.

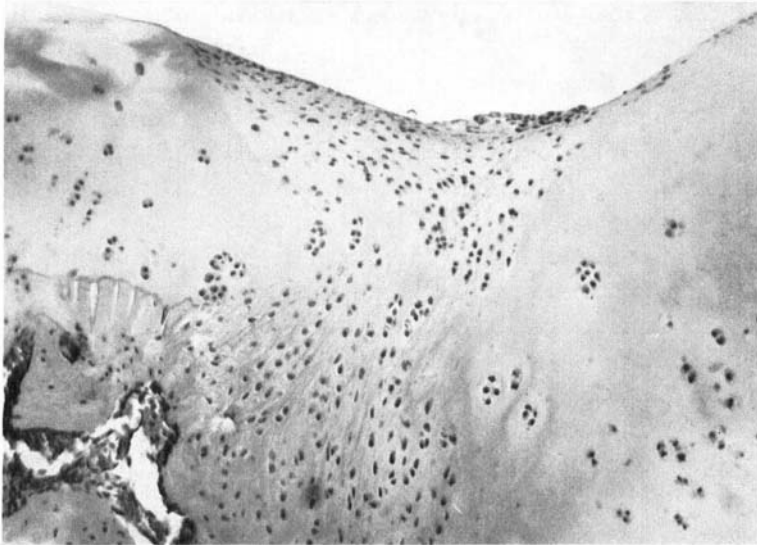


Figure 8. Junction of the cartilage of the bed with the cartilage of the graft 3 months after transplantation (64378-H). At the left the border of the necrotic cartilage of the bed, at the right the border of the necrotic cartilage of the homograft. In the centre of the picture there is cellular tissue with fine collagenous fibres. The groups of cells resembling the cells of hyaline cartilage can be seen in the isthmus. The fine collagenous fibres are oriented perpendicularly to the surface and near the surface parallel with it. Hematoxylin and eosin, $\times 45$.

DISCUSSION

During investigation of the development and ageing of traumatic pannus three basic stages can be distinguished:

1. In the first week, the *primary avascular pannus* is formed. It consists mostly of the network of fibrinous fibres among which numerous erythrocytes, polymorphonuclear leukocytes, histiocytic and fibroblastoid cells can be found. In view of the fact that mitoses of the latter cells were observed, one can take this type of the pannus for a tissue culture in a very advantageous intraarticular medium.

The pannus begins to develop from the first day after the lesion of the joint surface and fills all defects resulting from the damage of the articular cartilage. The extent and size of the pannus tissues not only depends on the adaptation of the graft or on the extent of the defect of articular surface, but also on the function of the joint. In this relation it makes no difference whether an autogenous or an homogenous graft was transplanted.

2. In the second and third week the original avascular pannus is gradually replaced by a *cellular fibrovascular pannus*. The source of this granulation tissue is the periosteum, synovial lining and subchondral marrow spaces. This tissue is characterized by a great number of fibroblastic cells, numerous blood vessels and fine collagenous fibres. This tissue replaces the whole avascular pannus and gradually matures into a fibrous tissue.

3. Starting with the fourth week after transplantation, the cellulofibrovascular pannus loses its original appearance. The number of cells and blood vessels decreases, whereas the bundles of collagenous fibres grow more numerous. Here and there islands of fibrocartilage or young bony tissue develop in the pannus. In this way a *final plastic pannus* is formed. In another case, mostly after the transplantation of homografts, resorption and defects occur in some parts of the graft. In such cases one can speak about a *lytic kind of a pannus*.

The formation of traumatic pannus not only influences the final shape of the articular surface, but also its quality. The extent of final pannus is given by the extent of primary avascular pannus. This means that if a minimal primary avascular pannus is formed, the final pannus will also be a minimal one. On the other hand, a large primary pannus will give rise to a large final pannus.

In all areas where the pannus has overgrown the articular cartilage, this cartilage necrotizes in its superficial layers or in the whole thickness. This necrotic tissue is replaced by connective tissue or fibrocartilage.

Therefore, it is important to decide when it is advantageous to support the formation of a traumatic pannus and when it is necessary to reduce its formation to a minimum.

In some experiments an effort was made, mainly after the lesion of the articular surface, to create conditions for the development of an intraarticular pannus. Krompecher (1967) removed the articular cartilage with a layer of the subchondral cancellous bone of the whole femoral portion of the knee joint and subjected such a joint to a functional load after the operation. He found that the granulation tissue, growing out from the subchondral spaces, was transformed into connective tissue with numerous islands of cartilage. Similar experimental arthroplasties were performed by Mooney & Ferguson (1966) and by Akeson and his associates (1969). In degenerative diseases of the joints drilling is proposed by Pridie (1959) and Insall (1967). The granulation tissue which grows out from the subchondral spaces covers the

articular surface to a limited extent and transforms into fibrocartilage. In our experiments (Bartoś & Fiala 1968) we tried to improve the conditions for the rearrangement of the osseous component of the homogenous osteocartilaginous graft by means of the drilling. The granulation tissue growing from the bed into the drilled holes in the graft acquired the character of a medullary pannus in the subchondral bone, as described by Rutishauser & Taillard (1966).

On the other hand, one tries to decrease the formation of a pannus. After transplantation of the articular surface or of the whole joint we attempted to secure early function of the joint by a perfect adaptation and firm fixation of the graft. In spite of early function of the joint, however, we were not able to keep the primary pannus down to a minimal extent. In the further course it is not possible to determine whether the cellular fibrovascular pannus will acquire a plastic or a destructive form.

It can be concluded that the quality of a transplanted articular surface depends not only on the reorganization of the subchondral bone, but also, to a certain degree, on the formation of the pannus. This will be a primary avascular pannus on the base of which a fibrovascular pannus and then a final plastic or lytic pannus may develop.

SUMMARY

Autogenous and homogenous osteocartilaginous grafts on 90 adult mongrel dogs with the aim of reconstructing the anterior non-weight-bearing portion of the distal end of femur were transplanted. The pannus, formed by fibrin and erythrocytes, filled in the first week after transplantation all the defects of the joint surface. The vascular granulation tissue grew into the primarily avascular pannus from the periosteum, synovial membrane or from the opened vascular spaces of the subchondral bone and changed its original structure. In the areas of blood vessel penetration a rich cellular population, mostly fibrocytes, and fine collagenous fibres could be found. This cellulofibrovascular pannus grew to maturity, the collagenous fibres became thicker, the cellularity and vascularity decreased and islands of fibrocartilage or osteoid tissue differentiated. The cartilage overgrown by the pannus necrotized in the superficial layers or in the whole thickness and was replaced by the connective tissue or fibrocartilage. At this time two forms of the pannus could be distinguished. The plastic one, composed of dense and strong collagenous fibres with a relatively small

amount of cells and vessels, formed the folds on the cartilage or bone of the graft and bed. The lytic one produced defects of the joint surface more in the graft than in the bed. The possibility of supporting or reducing the pannus formation in injured or diseased joints was discussed.

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