HISTOLOGICAL RESPONSE TO CARBON FIBRE

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The soft tissue response to carbon fibre was studied histologically one and a half years after being used to reconstruct the lateral collateral ligament of the human knee. A remarkably consistent pattern was seen in the induced ligament. The basic pattern was a "composite unit", consisting of a core of carbon fibre enveloped in a concentric manner by coherent layers of fibroblasts and collagen fibres. This new structure seemed to have been induced by continuous irritation caused by the physical structure of the carbon fibres; it is unlikely ever to acquire the structure of a natural ligament. However, it is biologically compatible and is biomechanically sufficient as long as the entire tow of carbon fibres is preserved.

For some years we have studied the host reaction to implanted carbon fibres (Mendes et al. 1983; Soudry et al. 1983). Encouraging results in experimental models and the absence of serious complications, as well as the reports of other investigators (Forster et al. 1978; Christel et al. 1980; Jenkins and McKibbin 1980; Aragona et al. 1981; Alexander, Weiss and Parsons 1983; Claes and Neugebauer 1983; Lemaire 1983; Neugebauer and Claes 1983; Strover 1983; Wolter 1983), have formed the basis for our clinical investigations.

This paper reports our observations on the histological features in patients one year or more after the implantation of carbon fibre.

MATERIALS AND METHODS

Since June 1981 we have operated on 45 patients for carbon fibre reconstruction of ligaments or tendons, in a variety of joints, for both acute and chronic injuries. In two patients the lateral collateral ligaments of the knee were completely excised one year and 18 months respectively after augmentation by carbon fibre. This was done during further attempts to improve the stability of the knee. These two ligaments form the material for the following histological study. The technique used at the original insertion of the carbon fibre tow strictly followed the principles described by us in a publication elsewhere (Mendes et al. 1985).

Carbon fibre. This was formed into a tow containing 40 000 carbon fibres (Plastafil) in two or three parallel but twisted bundles. The individual fibres had an average diameter of 8–10 μm and a breaking load of 0.15 N. The ultimate tensile strength of the complete tow was about 660 N. It was coated with biodegradable, medical grade gelatin to make it more resilient and to protect the fibres during operation. Special instruments were used to ensure careful and accurate surgical technique (Strover 1983).

Histology. The excised carbon fibre composite structure, or neoligament, was fixed in formalin for 48 hours, photographed and prepared for histology. The whole tow was embedded in paraffin. Both transverse and longitudinal sections were made, care being taken always to include the bed of the graft in the transverse specimens. Sections were cut 6 μm thick and stained, using: haematoxylin and eosin. Van Gieson and Masson techniques, reticulin stain, periodic acid Schiff technique with and without diastase, Weigert elastic stain, colloidal iron technique and Giemsa stain. Serial sections were prepared so that certain features could be followed over some distance.

Scanning electron microscopy. The composite ligament structure was removed from formalin, placed under vacuum for 12 to 24 hours and then dissected carefully to expose the carbon fibre tow. This was sectioned transversely and longitudinally, and gold-plated.

RESULTS

General structure. In no sections did we find the histological appearance of normal ligament or tendon, since the carbon fibre strands, the collagenous and reticulin fibres, and the fibroblasts and fibrocytes were always accompanied by inflammatory cells.

Under low magnification, it was possible to recognise the organisation of the cellular reaction to the
carbon fibre bundles which made up the tow. However, in most bundles, the central area was not yet organised, even when the peripheral and intermediate zones were organised. The cells participating in this were largely histiocytes, fibroblasts, fibrocytes and various leucocytes (Fig. 1).

**The composite ligament.** The appearance of the tow on cross section was especially impressive since individual carbon fibres were surrounded by two to four concentric layers of cells (Fig. 2). Most of these cells were fibroblasts and macrophages, with a minor admixture of lymphocytes, an occasional plasma cell and a very rare neutrophil. It was practically impossible, in routine histological sections, to distinguish between histiocytes and fibroblasts; we preferred to call them all histiofibroblasts. But in sections stained by Van Gieson, Masson or silver impregnation techniques the fibroblastic nature of these concentrically arranged cells was easily demonstrated by the formation of reticulin and also more mature collagen fibres (Fig. 3). In contrast to the abundance of collagen and reticulin fibres, no elastic fibres were found in the many sections studied except in the wall of blood vessels. In the longitudinal sections, the fibroblasts and the connective tissue fibres produced by them were noted to be consistently oriented in the direction of the carbon fibres with which they were associated (Fig. 4).

This organisation of fibroblasts and some other cells resulted in the formation of what we termed "units of the composite ligament". Each unit comprised a centrally located carbon fibre, surrounded by the cells which have been described above. These units were separated from each other by small amounts of loosely textured connective tissue, which was also infiltrated by a small number of leucocytes (Figs 2 and 3).

These features of the unit were also well illustrated in the scanning electron micrographs, and it was possible to demonstrate cells oriented along the intact carbon fibres. Two main types of cells were recognised. The first type was a relatively small, round and smooth cell, which according to Adams (1976) had the characteristic

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**Fig. 1**—Photomicrographs of organised tissue within a tow of carbon fibre. Figure 1—Longitudinal section (two carbon fibrils only are visible). Fibroblasts and fibrocytes are oriented parallel to the carbon fibrils, and there are also mononuclear and polynuclear macrophages and a few lymphoid cells (haematoxylin and eosin, × 150). Figure 2—Cross-section showing each carbon fibril encased in two to four layers of concentrically arranged histiofibroblasts. There are also a few lymphocytes and plasma cells (haematoxylin and eosin, × 200).

**Fig. 2**

**Fig. 3**—A silver-impregnated section of the same field as Figure 2. Each carbon fibril is surrounded by concentric layers of argyrophilic fibres (reticulum stain × 100).

**Fig. 4**—Longitudinal section to show a continuing inflammatory response in an otherwise well-organised carbon fibre tow. The histiofibroblasts adjacent to carbon fibrils are oriented parallel to them; at a distance from the fibrils the cells are arranged haphazardly. There are also many eosinophils and a few lymphocytes (haematoxylin and eosin, × 150).
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Scanning electron micrographs. Figure 5—Carbon fibres surrounded by small, round, smooth-surfaced immature macrophages and with histiofibroblasts (arrow) lying along them (× 1000). Figure 6—Stellate and fusiform histiofibroblasts and collagen fibres spreading out on and surrounding a few carbon fibres (× 470).

appearance of an immature macrophage, namely a monocyte. The second type of cell was stellate or fusiform; these cells were spread out with their processes along the carbon fibres, giving the impression that they were crawling on the carbon fibre (Figs 5 and 6).

Focal necrosis. There were a few small foci of necrosis, of various shapes, less than 2 mm in diameter and rather sharply demarcated (Fig. 7). These foci were easily seen in silver-impregnated sections because the argyrophilic fibres were absent in the necrotic area. It should be emphasised that no vascular or inflammatory changes which could possibly have caused the evolving necrosis were present in the many sections which were examined.

Synovial spaces. In a few of the many sections some irregular, fusiform spaces were seen (Fig. 8). These spaces appeared to be empty, but were lined by cells of synovial appearance (Fig. 9). The synovial-like spaces appeared between bundles of the carbon fibre tow which ran in different directions. The lining cells were cuboidal with abundant cytoplasm and roundish nuclei; fre-
sequently, these synovial-type cells were arranged in two or three layers. In addition, similar but elongated cells were occasionally seen in a palisade pattern. These appearances were reminiscent of those of a chronically inflamed synovial membrane.

**Do carbon fibres fragment?** Some of our experimental work (Mendes et al. 1983) showed that implanted carbon fibre tow does not fragment in vivo and, in fact, its satisfactory function probably depends on the preservation of the whole tow. Yet, as shown in Figure 8, fragments of carbon fibre were present in the sections when these were cut parallel to the direction of the fibres. Such fragments of carbon fibre were of various length; their edges were often sharp and it seemed that they were distributed in a haphazard fashion in the tissue section. In our opinion they were artefacts caused by fracture of carbon fibres during the sectioning of the paraffin blocks. Carbon fibre has inherent elastic energy and may therefore displace when cut. There is, however, no doubt that genuine breakage of carbon fibre also occurred in vivo, though rarely; carbon debris resulting from this was observed in mononuclear and polynuclear macrophages (see Fig. 9). The carbon debris phagocytosed by these cells appeared as tiny, irregular black granules within their cytoplasm.

**DISCUSSION**

At first sight it was surprising to find an active inflammatory response within a neo-tendon 18 months after implantation of a tow of carbon fibres. Although the fibroblasts which had invaded at an early period had synthesised collagenous and argyrophilic fibres in abundance, they were consistently accompanied by many macrophages, some lymphocytes, and a few plasma cells, neutrophils and eosinophils.

Our results therefore disagree with those of other investigators (Forster et al. 1978; Alexander et al. 1983; Wolter 1983). They have claimed that the implantation of carbon fibre tow has no untoward effect upon host tissue, and that the neo-ligament acquires the histological appearance of a natural ligament after a certain time interval. In our experience the inflammatory response does not subside, at least not for the first 18 post-operative months after insertion. Since the life span of the macrophages and various leucocytes is short (Adams 1976), it is self-evident that there is a continuous influx of monocytes, lymphocytes and granulocytes for a long period, possibly as long as the tow remains within the host. Once monocytes have migrated they mature into macrophages, and since these are facultative fibroblasts, they produce collagen and reticulin fibres. We believe that this transformation of monocytes into histiofibroblasts persists under the influence of the foreign material.

It should be noted that the histiofibroblasts, migrating between the carbon fibres rather than being haphazardly scattered (as they would be in non-specific granulation tissue), are consistently oriented along the carbon fibres. This being the case, one must be impressed by the orientation of the ingrowing fibrous tissue induced by the carbon fibres, especially as the collagen and reticulin fibres are also parallel to the carbon fibres.

It was surprising to find necrotic foci, since neither vascular lesions nor an infectious process were evident, and we believe that a toxic effect of carbon can be excluded. The possibility should be considered that the foci arise from circulatory compromise due to the relative motion and the varying compressive forces caused by the bundles of carbon fibre tow.

The synovium-lined spaces within the augmented tow are probably also related to the relative motion of carbon bundles one to another. It is known that abnormal motion of tissues relative to each other is associated with the formation of bursa-like structures, and we believe that a similar mechanism may be responsible for the formation of synovial spaces within the carbon tow. The observation that these spaces are characteristically found between bundles with different orientations supports our contention.

It has been suggested by other authors (Forster et al. 1978; Alexander et al. 1983; Wolter 1983) that the carbon fibres fragment continuously after implantation, that the fragments are removed by macrophages and that a new ligament is produced within months. The neoligament is said to consist of oriented connective tissue rich in collagen fibres which can replace the function of the carbon implant. Our findings do not support this mechanistic theory. The carbon fibres are not replaced within 18 months. On the contrary, they remain unchanged within the host and induce a foreign body reaction. The fragmented pieces of carbon fibre are, in our opinion, an artefact. Mechanical and material engineer colleagues at the Israel Institute of Technology (Brandon, personal communication) suggested, as mentioned above, that the displacement of the fragments was due to their inherent elasticity; a fibre cut under tension will relocate itself in unpredictable directions. Though the observed fragments are regarded as in vitro artefacts, there is no doubt that carbon fibres do break on rare occasions, and carbon debris is occasionally seen as tiny particles within mononuclear and polynuclear macrophages.

Our conclusion is that, although functionally adequate augmented ligaments and tendons can be induced by the implantation of tows of carbon fibre, the morphology of these is far from identical to that of the natural tissue. However, the association of carbon fibre with abundantly synthesised collagen and reticulin fibres which can function as replacement of the original ligament may be the important matter, rather than the failure to simulate its histology. Biomechanical analysis of the results of our animal experiments showed that the tensile strength of a composite tendon one year after insertion was about 85% of that of the natural one. The full results of these experiments will be published later (Mendes et al. 1985).
REFERENCES


