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A histological study on polyactive® for the prevention of peridural adhesions after spinal surgery: an experimental study in dogs with a 3 months follow-up

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In the present study the potential of Polyactive® bilayer sheets in the prevention of scar tissue formation after spinal surgery was investigated. Eight adult beagle dogs underwent 3 laminectomies at three levels (L2, L4 and L6). According to a randomized implantation schedule a Polyactive® sheet or autogenous fat graft was placed in the defect. The third site served as a control without implant. After 4 or 12 weeks (4 dogs per period) the dogs were sacrificed and histological sections were prepared. The Polyactive® treated defects showed partial closure by newly formed bone. The Polyactive® was encapsuled by a thin fibrous tissue layer. Ventral to the defect, dense fibrous tissue was present which was separated from the dura by the Polyactive® sheet in all cases. In some cases fibrous tissue was present between the implant capsule and the dura. In the fat graft group there were no signs of closure of the defect but most sites showed fibrous tissue at the edges of the graft, which was in 4 sites continuous with the dura mater. Fibrosis and degeneration of the fat grafts were seen. All control defects showed partial closure by newly formed bone, and ventral to the defect extensive fibrous tissue, which was in 50% continuous with the dura mater. Other sections showed loose connective tissue in contact with the dura mater. It is concluded that Polyactive® has a potential as a mechanical barrier in the prevention of adhesions between the dorsal spinal muscles and the dura mater.

1. Introduction

Various procedures in spinal surgery require access to the vertebral canal by laminectomy. After exposure of the dura mater, approximately 15% of the patients continue to suffer pain, which is well known as the "Failed Back Syndrome" [1]. Although it has never been proven that peridural fibrosis is the cause of the failed back syndrome [2], it has already been suggested by MacNab [3] that traction of the fibrotic tissue on the dura or nerve roots may cause low back pain or even pseudo-radicular pain. In addition, Benoist concluded in a study in human patients that 13 out of 38 patients had a beneficial result from dissection of scar tissue at reoperation [4]. Another aspect is that the existence of scar tissue after laminectomy makes it difficult to diagnose a new herniation of the disc, since interpretation of a CT-scan or MRI is difficult. Moreover, reoperation in the affected site is difficult with the risk of severe complications due to the fibrosis. Peridural fibrosis is the result from a healing process of the laminectomy site by fibroblastic activity. Several mechanisms responsible for this scar tissue formation have been described, such as (1) the invasion of fibroblasts from the damaged dorsal erector spinae muscles, forming the so-called 'laminectomy membrane' [5], (2) a result of a surgically damaged annulus fibrosis [6], and (3) organization of a local blood clot into fibrotic tissue [2].

Many investigators performed studies to prevent postlaminectomy sear tissue formation. These studies either used a mechanical barrier [7–11] aiming at the prevention of adhesion formation to the dura mater/nerve roots, or used a chemical agens that influenced the process of scar tissue formation [2, 12–16]. The majority of these barriers or agens have never reached a clinical application, despite the promising results shown by some of them. The only material that is now used in clinical medicine by some surgeons is a biological mechanical barrier, composed of an autogenous free fat transplant [17–19]. However, the use of a fat graft cannot prevent scar tissue formation in all situations and complications such as

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necrosis, fibrosis and spinal cord compression due to migration of the fat body into the epidural space have been described [20].

Recently, it has been suggested that a degradable polymer called Polyactive® might have a potential as a mechanical barrier to prevent adhesions to the dura mater after laminectomy. This is based upon experiments in rats that showed that Polyactive® bilayers significantly reduced postoperative intraperitoneal scar tissue formation [21]. Intra-abdominal studies are currently performed in human patients and in a pilot experiment in dogs the potential of Polyactive® as a mechanical barrier was indeed confirmed [11].

In the present experiment, the potential of Polyactive® bilayer sheets in the prevention of scar tissue formation after spinal surgery was further investigated. In particular, the histological response to Polyactive® was evaluated and compared with a free fat graft and a control.

2. Materials and methods

2.1. Polyactive®

Spinal barrier sheets were made of 1000PEG60PBT40 Polyactive® (also referred to as PEO/PBT copolymer) by HC Implants B. V. Leiden, The Netherlands, using a solvent casting method. The resulting bilayer consisted of a dense top layer and a porous under layer and had a total thickness of approximately 280 μm. After casting and chloroform evaporation, the films were immersed in distilled water to rinse out the salt and were air dried. Barrier sheets (50 mm × 50 mm × 280 μm) were cut from the film, vacuum-sealed and sterilized by gamma-irradiation at ≥ 25 kGy. The structure of the bilayer was evaluated by scanning electron microscopy (Fig. 1). During surgery, the Polyactive® sheets were trimmed with a pair of scissors to fit the defect site. The sheets were positioned ventral to the laminectomy defect with the dense side facing the dura mater.

2.2. Study design

The present study was a prospective, randomized, cross-sectional and cohort study in experimental Beagle dogs. Three treatments for the prevention of post-laminectomy scar formation were compared: (1) Porous/dense sheets made of 1000PEG60PBT40 Polyactive®, (2) free autogenous fat grafts and (3) control laminectomy without placement of Polyactive® or fat graft. A total of 8 adult dogs were used in the experiment. In each animal, three laminectomies were performed at the lumbar vertebrae L2, L4, and L6. The three treatments were allocated to three laminectomy sites at the lumbar spine, according to a randomized implantation schedule (Table 1). This schedule was designed to neutralize a possible influence of small differences between the three laminectomy levels and provided auto-controls within each animal. The animals were allocated to two follow-up periods (four animals per period) of four and 12 weeks.

TABLE I Summary of implantation schedule. C = control; P = Polyactive®; F = fat graft

Animal no.	Follow-up (weeks)	Treatment *L2*	*L4*	*L6*
B24-94	4	C	P	F
B25-94	4	C	F	P
B27-94	4	F	P	C
B28-94	4	P	C	F
B29-94	12	F	P	C
B31-94	12	C	F	P
B32-94	12	P	F	C
B33-94	12	P	C	F

2.3. Animals

Eight healthy adult beagle dogs of both genders, weighing 11–15 kgs, were obtained from a professional stock-breeder. The dogs were housed individually in indoor kennels under a normal daylight regimen. They were fed a commercially dry, pelleted dog food (D. B. Brok, Hope Farms BV, Woerden, The Netherlands.) and they had free access to water. The animals were adapted to these conditions from two weeks prior to the start of the experiment. The dogs were examined by a veterinary orthopaedic surgeon (BM) prior to surgery. Surgery was performed after an overnight fast.

2.4. Anaesthesia

Following intramuscular premedication with 0.1 mg acepromazine (Vetranquil®, Sanovi BV, Paris, France.)/kg, 1 mg methadone (Symoron®, Gist-Brocades NV, Delft, The Netherlands.)/kg, and 0.1 mg atropine (Atropini Sulfas®, Pharmachemie BCV, Haarlem, The Netherlands.)/kg, anaesthesia was induced by intravenous administration of 10 mg sodium penthotal (Nesdonal®, Rhône Merieux, Lyon, France.)/kg. The dogs were intubated, and inhalation anaesthesia was maintained in a semi-closed system with a mixture of Halothane (Halothane, Albic BV, Maassluis, The Netherlands.), NO_2 and oxygen. Preoperative antibiotic prophylaxis consisted of intravenous administration of 3 mg gentamicin (Gentamicin 5%, AUV, Cuijk, The Netherlands.)/kg and 5 mg lincomycin (Lincocin®, Upjohn NV, Ede, The Netherlands.)/kg. To minimize spinal cord edema due to surgical trauma, 1 mg dexamethasone (Dexadreson®, Intervet International BV, Boxmeer, The Netherlands.)/kg was administered intravenously prior to surgery.

2.5. Surgical technique

The lumbar and lumbosacral region were clipped and the surgical field was disinfected with povididone-iodine (Betadine®, Dagra, Diemen, The Netherlands.). The laminae of lumbar vertebrae L6, L4, and L2 were successively approached through a dorsal incision as described previously [22]. The lumbar fascia and supraspinous ligament were incised around each spinous processus, and on the midline between each

processus. Elevation was most easily done from a caudal-to-cranial direction. Dorsal laminectomy was started by removal of the spinous processus with a rongeur and the lamina with a pneumatic burr under continuous irrigation and cooling with saline solution. Burring was discontinued when the ventral cortical layer of the vertebral lamina was encountered. After careful incision of the interarcuate ligament, the remaining laminar cortical layer was removed using fine neurosurgical punches, thus minimizing surgical trauma to the dura mater and spinal cord. The dura mater was exposed by carefully removing the interarcuate ligament and the epidural fat. The laminectomy defect created in this way measured 5 by 20 mm for each site. According to the implantation schedule, one of the three treatment options was applied (Polyactive®, fat, control). In case an autogenous fat graft was allocated to the laminectomy site, this was obtained from the subcutaneous fat dorsal to the surgical site. After finishing the treatment of the L6 region, the multifidus lumborum muscle was replaced into its original position and the lumbar fascia was partially closed with interrupted adsorbable polyglactine (Vicryl®, Ethicon, Norderstedt, Germany.) sutures.

Postoperative analgesia was provided by intravenous administration of 0.1 mg buprenorphin (Temgesic®, Shering-Plough, Reckitt & Coleman Products, England.)/kg at the end of surgery and subsequently subcutaneously every eight hours for at least 24 hours. The animals were observed carefully in the postoperative period, and if additive pain relief was necessary, buprenorphin administration was prolonged. Postoperative antibiotic prophylaxis consisted of subcutaneous administration of 3 mg gentamicin/kg and 5 mg lincomycin/kg every eight hours for 24 hours. Subsequently, 12.5 mg amoxycillin/clavulanic acid (Synulox®, Smithkline Beecham Animal Health BV, Zoetermeer, The Netherlands.) /kg BID was given orally for seven days.

Protocols for all experiments involving the use of dogs were approved by the Ethical Committee for animal experiments of the Faculty of Medicine, Utrecht University, The Netherlands.

2.6. Retrieval and analysis of implant-tissue blocs

At the end of the follow-up periods the animals were euthanatized with a concentrated pentobarbital solution (Dolethal®, Vétoquinol BV, 's-Hertogenbosch, The Netherlands.). Cerebrospinal fluid (CSF) was collected through a puncture in the neck. CSF samples were analyzed with regards to cytology, glucose and protein content. In addition peripheral blood samples were analyzed for glucose content. All fixation and dehydration procedures were performed at room temperature. The distal spine (L1–L7), including the intrinsic musculature, was taken out *en bloc*, and placed in a buffered formaldehyde solution for at least three days. Then, using a band saw, the L2, L4 and L6 levels were sawn out such that the entire laminectomy site was taken out without damage. These tissue blocks were again placed in a buffered formaldehyde solution. After one week, they were sawn transversally into two halves, through the middle of the laminectomy site, and replaced in a buffered formaldehyde solution for final fixation, and dehydrated in alcohol series. The proximal half was embedded in methyl-methacrylate (MMA), which was allowed to polymerize (PMMA). Subsequently, sections of 5–10 μm thickness were cut from the distal side using a Jung-K microtome. These sections were mounted on gelatine-coated slides, and the PMMA was removed using 2-methoxyethyl-acetate. Then the sections were recoated with celloidine, and stained according to Goldner. Of the remaining proximal sample 5–10 μm sections were cut using a specially designed hard tissue saw [23]. These sections were stained with basic fuchsine/ methylene blue. A slice of the distal half of the tissue bloc was decalcified, embedded in paraffin and thin routine pathology sections were cut and stained with haematoxilin and eosin.

The laminectomy and peridural sites of all sections were examined with a light microscope (Olympus BX-50) to judge the tissue response to the various treatments, the fate of the laminectomy defect, and the extent and morphology of scar tissue formation. The organization of the collagen fibres was judged using polarized light.

3. Results

3.1. General

The animals recovered quickly from surgery without any complications. Within one day after surgery, all animals were standing up and walking again. During the post-operative follow-up period, none of the animals developed neurological deficit. The results from the cerebrospinal fluid and blood samples are presented in Table II. Macroscopical inspection of the transverse sections through the L2, L4 and L6 laminectomy sites did not reveal gross pathology.

3.2. Histology

3.2.1. Polyactive®

The laminectomy defects showed partial closure by newly formed bone (four weeks up to approximately 60%; 12 weeks up to approximately 95%). Bone formation was visible both as enchondral ossification and as osteoid deposition by osteoblasts directly on the new bone at the defect edges. One 12 weeks-sample showed no signs of closure of the laminectomy defect. The central, non-closed zone of the defect consisted of fibrous or fibrocartilagenous tissue, which continued ventrally as a dense fibrous tissue layer with orientation of the collagen fibres parallel to the dura mater/Polyactive® sheet (Fig. 2). The Polyactive® sheet was surrounded by a fibrous tissue capsule of 5–25 cell layers. At the ventral side of the sheet, between the capsule and the dura mater, all four weeks samples showed a fibrous tissue interlayer with a thickness between approximately 20 to 50 cells (Fig. 2). The density of this layer was high, but not as dense as the fibrous tissue dorsal from the Polyactive®. This fibrous tissue interlayer was also seen in two of

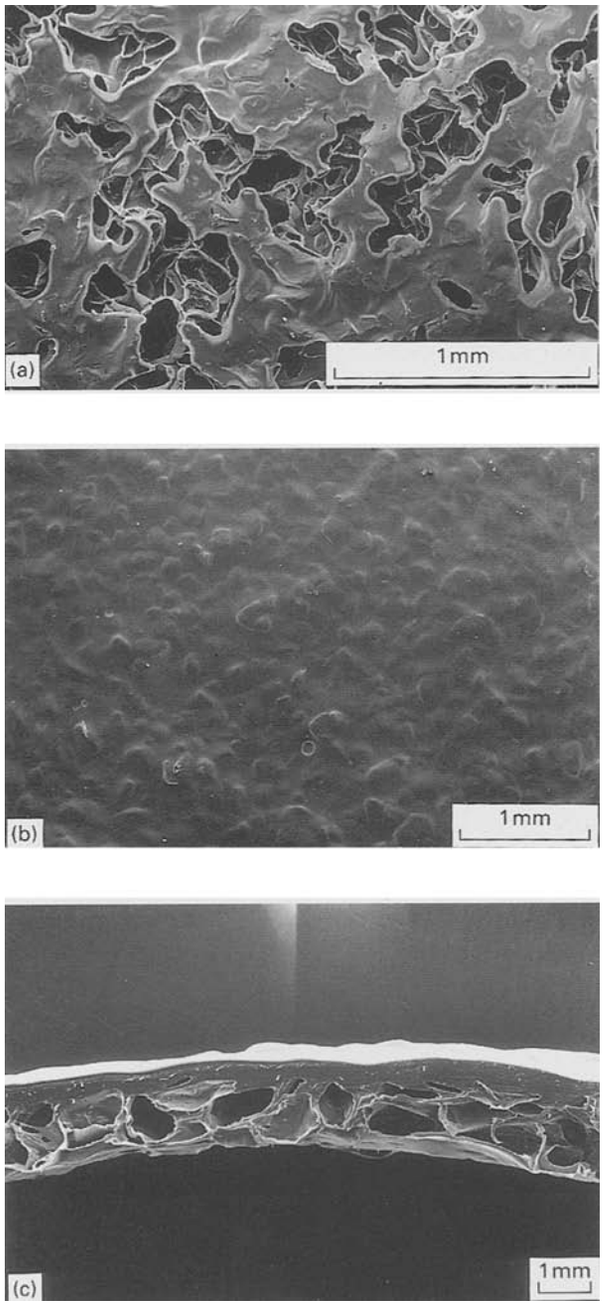

Figure 1 Scanning electron micrograph of Polyactive® barrier sheet showing porous (a), dense (b), and lateral (c) views.

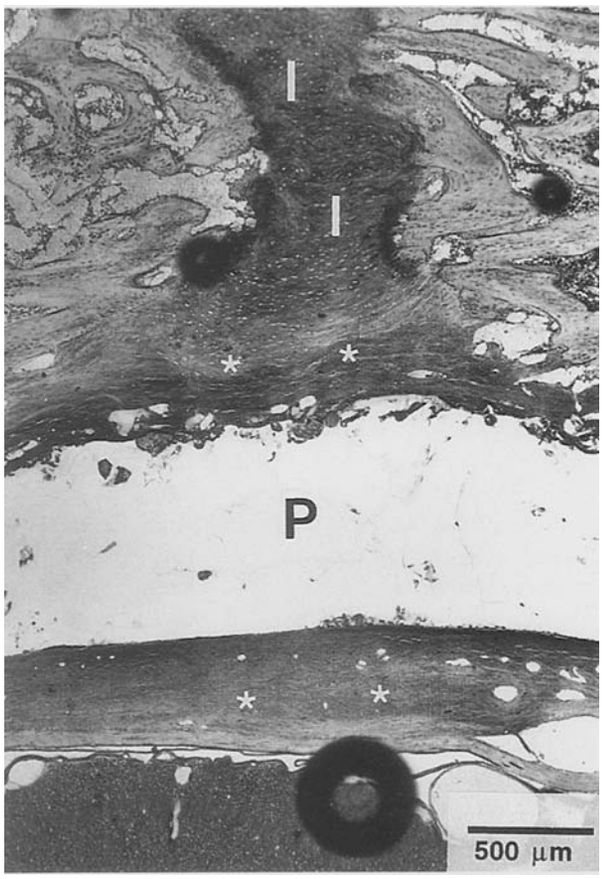

Figure 2 Light micrograph of laminectomy defect treated with Polyactive® barrier sheet, 4 weeks after implantation. Closure of the laminectomy defect (1) is visible with a central zone of fibrocartilagenous tissue. Fibrous tissue (*) is present ventral and dorsal to the implant (P). Nondecalcified section stained with basic fuchsine/methylene blue.

the 12 weeks samples. The other two samples did not show fibrous tissue between the Polyactive® capsule and the dura mater (Fig. 3). As mentioned above, the local tissue response to the implant consisted of a thin fibrous tissue capsule at both follow-up periods. However, at the porous side of the sheet, a more active tissue response was seen than at the smooth side represented by predominantly macrophages and sometimes multinuclear giant cells. It was difficult to judge the degradation of the Polyactive®, since during histological processing the specimens might have been damaged. Both at four and 12 weeks, the thickness of the sheet did not seem to be affected, but especially at 12 weeks cracks were seen in the material. After four weeks, irregular Polyactive® fragments were present along the porous side and at the edges, surrounded by

TABLE II Results from cerebrospinal fluid and blood samples. Normal values are: erythr. none; leuc. < 5 c/mm^3; protein < 10–30 mg/dl; liq.gluc. $< 70\%$ of bl.gluc.

Animal no.	Follow-up (weeks)	erythr. (c/mm^3)	leuc. (c/mm^3)	protein (mg/dl)	liq.gluc. (mmol/l)	bl.gluc. (mmol/l)
B24-94	4	3	1	< 10	3.1	4.3
B25-94	4	3413	26	24	2.1	4.7
B27-94	4	193	1	15	3.2	4.4
B28-94	4	635	1	12,4	3.2	4.4
B29-94	12	3	1	< 10	3.1	4.3
B31-94	12	137	12	20	2.9	4.5
B32-94	12	1	1	< 30	3.6	4.3
B33-94	12	0	1	24	3.3	5.1

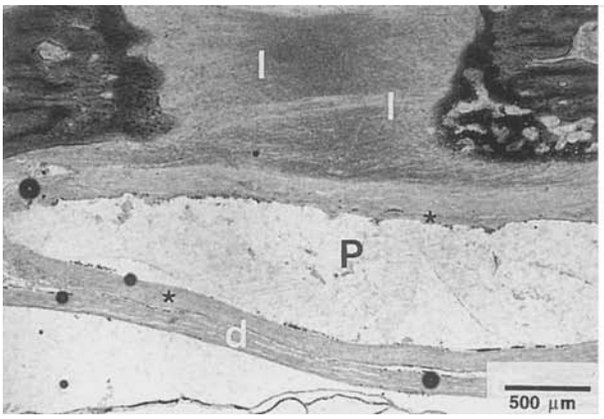

Figure 3 Light micrograph showing the Polyactive® sheet (P), 12 weeks after implantation, surrounded by a thin fibrous tissue capsule (*). No other fibrous tissue is present in-between the capsule and the dura mater (d). The laminectomy defect (1) is filled with fibrous tissue. Nondecalcified section stained with basic fuchsine/methylene blue.

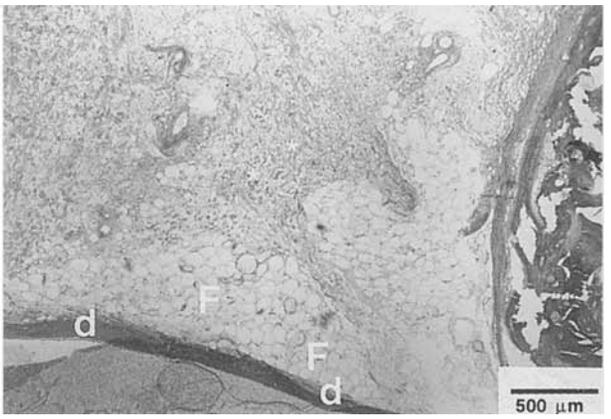

Figure 5 Light micrograph of laminectomy treated with a fat graft, 4 weeks after surgery. The lateral edges of the laminectomy defect do not show bone formation as a sign of defect closure. The dura mater (d) is covered with a zone of vital fat tissue (F), but more towards dorsally degeneration and fibrosis are present (*). Nondecalcified section stained with basic fuchsine/methylene blue.

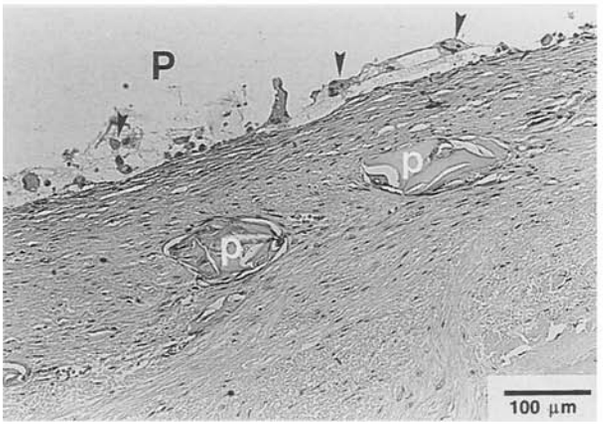

Figure 4 Detail of dorsal side of Polyactive® (P), facing the laminectomy defect, 12 weeks after implantation. Polyactive® (p) fragments are surrounded by macrophages and fibroblasts and cells with a 'foamy' cytoplasm are visible (arrowheads) at the implant surface. Decalcified section stained with heamatoxilin and eosin.

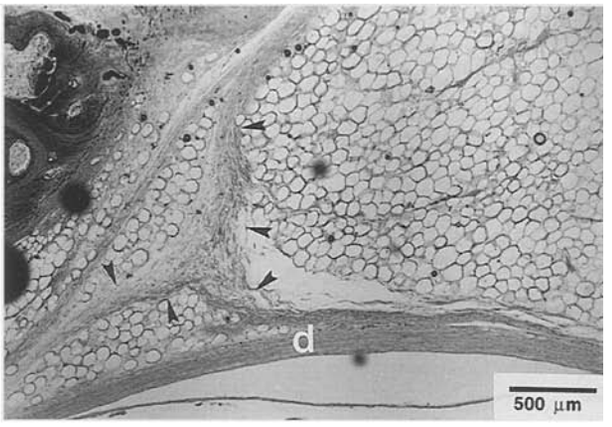

Figure 6 Light micrograph of laminectomy treated with a fat graft, 12 weeks after surgery. Strings of fibrous tissue (arrowheads) are present in the fat graft and sometimes continuous until the dura mater (d). Nondecalcified section stained with basic fuchsine/methylene blue.

macrophages and/or multinuclear giant cells. This was also seen after 12 weeks, but these specimens also showed cells with single intracellular fragments and macrophages with a 'foamy' cytoplasm (Fig. 4). Both the porous and dense sides of the sheet showed a slightly undulating aspect after 12 weeks and the porous side could not easily be differentiated from the dense side. After 12 weeks, two sheets seemed to have migrated laterally, but still the original defect was fully covered. One sheet was fold double at the edges, but this did not affect the local tissue response.

3.2.2. Fat grafts

None of the laminectomy defects treated with a fat graft showed signs of (partial) closure, both after four and 12 weeks. The only bone formation present consisted of osteoid deposition on the lateral original edges of the defect. Three of the four weeks sites revealed that close to the dura mater a zone of vital, non-fibrotic fat tissue was present, but more dorsally, the fat graft showed increasing fibrosis and areas of degeneration (Fig. 5). Strings of fibrous tissue were present in the fat graft and sometimes continuous until the dura mater (Fig. 6). It could not be differentiated whether these strings were pre-existing or newly formed. One four weeks defect showed a dense fibrous tissue layer onto the dura mater, continuous with fibrous tissue lateral to the fat graft in the laminectomy defect. After 12 weeks, the fat graft could still easily be detected. Three defects showed fibrous tissue next to the fat grafts (in-between the graft and the defect edges) which extended towards the dura mater (Fig. 7). However, fat cells were usually in contact with the dura mater. One sample showed that the fat graft was separated by a dense fibrous tissue plate covering the defect and another sample showed that the ventral side of the defect was covered by fibrous tissue which was in contact with the dura mater.

3.2.3. Controls

All control defects showed partial closure of the defect by newly formed bone (four weeks up to approximately 65%; 12 weeks up to approximately 95%). Bone

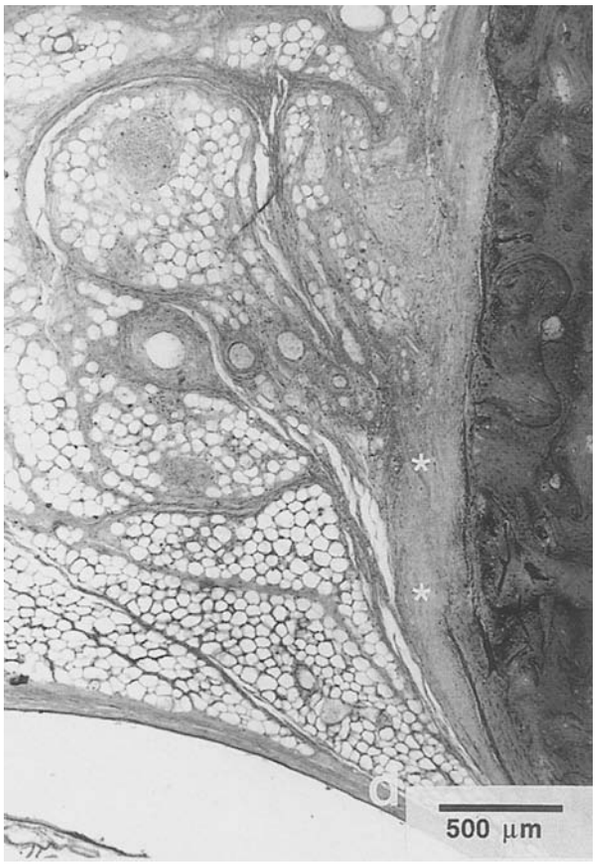

Figure 7 Light micrograph of fat graft, 12 weeks after implantation. Fibrous tissue (*) is present next to the graft and extends in the direction of the (lateral) dura mater (d). Nondecalcified section stained with basic fuchsine/methylene blue.

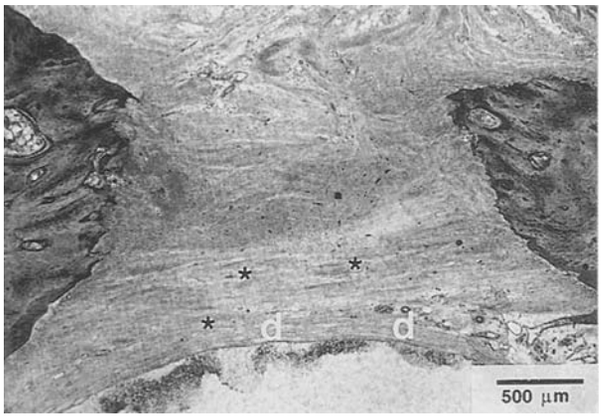

Figure 8 Light micrograph of control defect, 12 weeks after surgery, showing a dense fibrous tissue layer (*) extending from the laminectomy defect until the dura mater (d). Nondecalcified section stained with basic fuchsine/methylene blue.

formation was visible both as enchondral ossification and as osteoid deposition by osteoblasts directly on the new bone at the defect edges. One 12 weeks defect did not show any closure of the defect and was filled with fibrous and fat tissue. Ventral to the laminectomy defect all control sites showed a dense fibrous tissue layer which in 50% of the specimens (both four and 12 weeks) was continuous with the dura mater (Fig. 8). The other specimens showed that in the proximity of the dura mater the aspect of the fibrous tissue layer

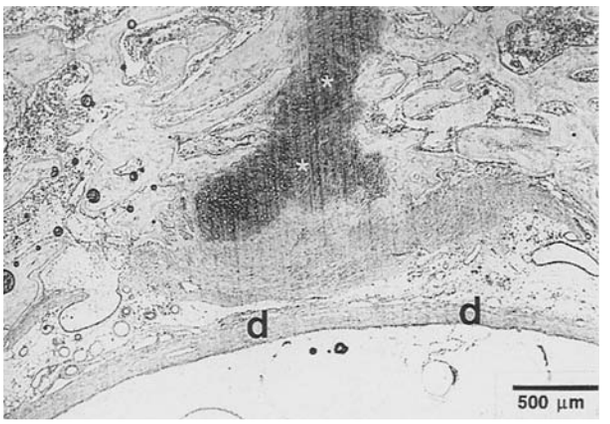

Figure 9 Light micrograph of control defect, four weeks after surgery, showing partial closure of the defect. Central in the defect a zone of fibrocartilagenous tissue (*). Towards the dura mater (d) dense fibrous tissue is visible, but close to the dura, loose connective tissue is present. Nondecalcified section stained with basic fuchsine/methylene blue.

changed into loose connective tissue with fat cells and blood vessels (Fig. 9). The orientation of the collagen fibres in the dense fibrous tissue was predominantly parallel to the dura mater, but also other fibre directions were seen. The organization of these collagen fibres was less uniform when compared with the fibrous tissue dorsal to the Polyactive® sheets.

4. Discussion

The formation of scar tissue is a normal wound healing process and usually in favour of both patient and surgeon. However after a laminectomy, the localization of the scar tissue can give problems. The suggestion that epidural scar tissue formation is clinically related to low back pain, sciatica and radicular pain is confirmed by findings that scar tissue excision reduces the symptoms [4, 24]. The best approach would be to prevent the epidural scar formation to grow in such a way or to such an extent that it can give clinical complaints. In the current investigation, we used a laminectomy model in the dog, in which the dura mater was exposed, to describe the scar tissue formation after three different treatments. A new mechanical barrier, Polyactive®, was compared with the current 'golden standard' (fat graft) and a sham-operated control; all three treatments were applied to each dog. The rationale for a mechanical barrier is based upon the findings by LaRocca [5] that fibrosis is a result from the migration of fibroblasts from the dorsal musculature towards the dura. Interposing material between the dorsal musculature and the dura could prevent adhesions to the dura. Other treatment options include those that aim at the reduction of hematoma formation [19] or at the reduction of fibroblastic activity itself [2, 8, 12–16]. The rationale for the use of a fat graft by various investigators is probably based upon its efficacy as a mechanical barrier, but also upon the theory that because of the space occupying dimensions of the graft, a hematoma cannot be formed in the area where the graft is positioned.

In the present study, the slightly elevated leucocyte count in two samples is explained by the presence of erythrocytes, suggesting addition of blood to the punctate. Although these tests do not differentiate between the three treatment options, they suggest that intrathecal inflammation was not present. We confirmed that one possible mechanism responsible for the peridural adhesions and scar formation is migration of fibroblasts from the posterior musculature. Both the controls and the Polyactive® treated defects showed fibrous tissue in the laminectomy defect extending ventrally. However, both groups also showed frequent closure of the laminectomy defects, but still fibrous tissue was present ventral to the new lamina. Fifty percent of the control defects showed ventral to the defect a dense fibrous tissue layer continuous with the dura. It seems obvious that when a mechanical barrier can prevent this continuity of scar tissue with the dura mater, clinical symptoms may be reduced. The Polyactive® membrane used in this study showed to be an effective mechanical barrier up to 3 months after surgery. All Polyactive® treated defects showed scar tissue formation dorsal to the implant resembling the control defects, but as a result of the presence of the implant, this scar tissue formation never continued to the dura. The Polyactive® was surrounded by a thin fibrous tissue capsule indicating a very mild tissue response. This is comparable to previous studies on the intra-abdominal application of Polyactive® [21]. Ventral to the Polyactive® frequently a fibrous tissue interlayer between the capsule and the dura was seen. Despite adequate hemostasis and flushing with saline, blood still can flow between the dura and the Polyactive® after placement of the membrane. The formation of this fibrous tissue interlayer is explained by the organization of blood cells into fibrous tissue. However, since this fibrous tissue interlayer was never continuous with the scar tissue dorsal to the implant, it is not likely that it can act as a mediator for traction to the dura.

Polyactive® has been described as a biodegradable product [25]. After 12 weeks, the membrane was still present, but signs of degradation of the material were obvious. Crack formation, which was previously described as a sign of degradation [25], was seen, and small Polyactive® fragments were frequently detected at the porous side of the material. These fragments were usually surrounded by macrophages and/or multinuclear giant cells, and sometimes also intracellular fragments were seen. It is not yet known what will occur after the Polyactive® membrane has degraded. It can be speculated that since the continuation of the dorsal fibrous tissue to the dura was interrupted, this will remain the end-state showing that Polyactive® is an effective barrier. On the other hand, it is also possible that the Polyactive® is replaced by fibrous tissue and that the material just delayed the process as described for the sham-operated control group.

Since 1965 free fat transplants placed on the spinal dura after surgery on lumbar discs have been investigated in many experiments [10, 19, 26]. In all defects treated with a fat graft in the current study, we found the defects to remain open. We considered this an unfavourable situation since so the myelum remains more vulnerable to dorsal traumata. In addition, it has been described that lamina repair with a solid material covering the exposed dura significantly reduces the formation of scar tissue [7, 8]. With respect to the prevention of scar tissue formation by the fat grafts, we found already after 4 weeks signs of fibrosis and degeneration of the dorsal part of the fat graft. Ventrally usually vital fat cells were in contact with the dura. After 12 weeks in most defects fibrous tissue was present next to the grafts and extending towards the dura. These findings suggest that the fat grafts are vulnerable to fibrosis or replacement by other tissues and that their scar-preventing potential is limited.

In the present study, it was confirmed that Polyactive® has a potential as a mechanical barrier in the prevention of adhesions between the spinal muscles and the dura mater. It is likely that continuation of scar tissue formation is prevented by the presence of the Polyactive® sheet. Studies using longer follow-up periods are currently under investigation in our laboratory.

Acknowledgements

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