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Biloshytskyi R., Kalachniuk L., Grushanska N., Bokotko R.,
Dymko R., Tkach G., Kladnytska L., Shupyk O., Rodz V., Fedyshyn P.

NEUROLOGICAL SYNDROMES AFTER TRAUMA IN DOGS (ETIOLOGY, SYMPTOMS AND TREATMENT)

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Authors:
R. Biloshytskyi, L. Kalachniuk, N. Grushanska, R. Bokotko, R. Dymko,
G. Tkach, L. Kladnytska, O. Shupyk, V. Rodz, P. Fedyshyn

Reviewers:
O. Savchuk, Doctor of Biological Sciences, Professor, Head of the Department of Biochemistry, ESC "Institute of Biology and Medicine" of Taras Shevchenko National University of Kyiv;
B. Gutyj, Doctor of Veterinary Sciences, Professor, Head of the Department of Hygiene, Sanitation and General Veterinary Prevention of Stepan Gzhytskyi National University of Veterinary Medicine and Biotechnologies of Lviv;
S. Golopura, Doctor of Veterinary Sciences, Professor, National University of Life and Environmental Sciences of Ukraine.

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The monograph is devoted to the study of the pathogenesis, clinical presentation, and effective approaches to the diagnosis and treatment of traumatic neurological injuries in dogs. Special attention is given to a comprehensive approach to identifying neurological syndromes resulting from trauma using clinical, neurological, laboratory, and instrumental diagnostic methods.

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e-mail: *editor@promonograph.org*
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ABOUT THE AUTHORS:

Roman Biloshytskyi, PhD in Veterinary Sciences, National University of Life and Environmental Sciences of Ukraine; https://orcid.org/0000-0003-3753-1748

Liliia Kalachniuk, Doctor of Biological Sciences, Professor, Professor of the Department of Biochemistry named after Acad. M.F. Gulyi National University of Life and Environmental Sciences of Ukraine; https://orcid.org/0000-0002-5545-8495

Nataliia Grushanska, Doctor of Veterinary Sciences, Professor, National University of Life and Environmental Sciences of Ukraine, https://orcid.org/0000-0001-8447-2758

Roman Bokotko, PhD in Veterinary Sciences, Associate Professor, National University of Life and Environmental Sciences of Ukraine, https://orcid.org/0000-0002-6217-5266

Roman Dymko, PhD in Veterinary Sciences, Associate Professor, Associate Professor of the Department of Animal and Food Hygiene named after professor A. Skorokhodko at the National University of Life and Environmental Sciences of Ukraine, https://orcid.org/0000-0003-4135-5310

Gennadii Tkach, Doctor of Medical Sciences, Professor, National University of Life and Environmental Sciences of Ukraine; https://orcid.org/0000-0002-6482-4792

Larysa Kladnytska, Doctor of Veterinary Sciences, Professor, National University of Life and Environmental Sciences of Ukraine, https://orcid.org/0000-0002-9360-0587

Oleksandr Shupyk, PhD in Veterinary Sciences, Associate Professor, National University of Life and Environmental Sciences of Ukraine; https://orcid.org/0000-0002-1081-3932

Vladyslav Rodz, Doctor of Veterinary Medicine, "Kyiv Vet Podil", https://orcid.org/0009-0008-3614-5435

Petro Fedyshyn, PhD student, National University of Life and Environmental Sciences of Ukraine, https://orcid.org/0000-0002-3706-2788

CONTENT

LIST OF SYMBOLS, SYMBOLS, UNITS, ABBREVIATIONS AND TERMS

C1 - C5 - vertebrae of the cervical spine

Cd1 - Cd5 - vertebrae of the caudal spine

EDTA - ethylenediaminetetraacetic acid

HU - Hounsfield Units

IVDD-1 - Hansen type I intervertebral disc disease

IVDD-2 - Hansen type II intervertebral disc disease

L1 - L5 - vertebrae of the lumbar spine

M - arithmetic mean

m - error of the arithmetic mean

PLT - platelets

S1 - S3 - vertebrae of the sacral spine

T1 - T7 - vertebrae of the thoracic spine

UMN - upper motor neuron

IVDD - degenerative diseases of the intervertebral disc

DSO - decompression and stabilisation operation

Disc displacement - displacement of the intervertebral disc

CT scan - computer tomography scan

IVD - intervertebral disc

MR - magnetic resonance imaging

LMN - lower motor neuron

LSR - lumbosacral region

SC - spinal cord

SAP - subarachnoid space

CSF - cerebrospinal fluid

SCI - spinal cord injury

CNS - central nervous system

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INTRODUCTION

Relevance of the topic. Injuries to the spine and spinal cord of dogs are one of the most common diseases of this species, especially in urban areas. They cause suffering to animals and their owners and often result in death. The relevance of proper diagnosis and treatment of spinal diseases is increasing with the expansion of the arsenal of the latest research methods. This, in turn, makes it possible to identify new patterns of pathological processes, establish their causes and apply modern, more effective methods of prevention and treatment.

Determining the exact location and degree of brain damage allows you to correctly establish the diagnosis and determine the necessary methods of conservative or surgical treatment. It is important that the diagnosis is carried out in a short time, as timely stopping the development of the inflammatory process within the dense tissues of the spinal canal is often critical.

Numerous studies by scientists from many countries have confirmed the widespread prevalence of spinal cord and spinal cord injuries in dogs. The issues of diagnosis and treatment of diseases of the nervous system and spine have been studied at different times and developed in the works of Curtis W. Dewey, 2003-2022 (USA) [50, 51, 52]; Driver C.J. et al., 2013 (UK) [56]; Takagi S. and collaborators, 2022 (Japan) [58]; Sands J. et al., 2022 (USA) [62]; Cho W. and collaborators, 2022 (USA) [65]; Antończyk A. & Ochota M., 2022 (Poland) [69]; Adami C. & Gendron K., 2017 (UK & USA) [70]; de Lahunta A., 2008 (USA) [73].

Over the past decades, radiological and tomographic diagnosis of spinal cord and spinal cord injuries has been studied and implemented in clinical practice in the different countries – Rohdin C. et al., 2020 (Sweden) [74]; Kealy J. K., McAllister H., 2000 (USA) [77]; Dennis R., Kirberger M.R., Barr F, 2010 (USA) [79]; Easton S., 2010 (USA) [80]; Andersen-Ranberg E., Berendt M., Gredal H., 2021 (Denmark) [89]; Landsgaard K.A. et al., 2023 (USA) [90]; Kim U. et al., 2025 (South Korea) [95]; Paninárová M. et al., 2016 (Czech Republic)[96]; Sukhonos V.P. & Biloshytskyi R.V., 2018 (Ukraine) [98]; Sakata A. et al., 2025 (Japan) [75]; Rusbridge C., 2017 (UK)

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according to [99] and others.

Insufficient attention has been paid to the diagnosis and treatment of spinal cord and spinal cord injuries, for insctsnce, publication of Ukrainian scientists, as: Sukhonos V. P. [97], Doroshchuk V.O. & Sokol A.I. [106], Skrypka, M. et al. [107] etc. The biochemistry of nervous tissue was also studied by Ukrainian scientists, as: O. V. Palladin, G. E. Volodymyrov, E. M. Kreps, Y. V. Belik [108], their schools and followers.

Neurological deficits in dogs, manifested in the form of paresis and limb paralysis, are often caused by spinal cord compression due to impaired blood circulation and edema formation. Minor spinal cord injuries sometimes do not have a clinically evident neurological deficit, while partial or complete rupture of the spinal cord causes spinal shock and ascending myelomalacia syndrome with a poor prognosis. A detailed study of neurological syndromes in combined spinal cord injury contributes to the correct choice of conservative and surgical treatment methods in dogs with a course of postoperative rehabilitation.

The use of hemilaminectomy, mini-hemilaminectomy, fenestration and foramenectomy is advisable because they are less invasive than other methods in pathological changes of intervertebral discs. Surgical methods of treating spinal cord and spinal cord injuries are not sufficiently covered in the literature. All this necessitates the search for new, more effective methods of treatment and diagnosis of spinal injuries in dogs.

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SECTION 1
LITERATURE REVIEW

1.1. Structure and function of the dog spine

The spine is one of the most important structures in the body. It is an organ of support and movement, plays the role of a link between the head, shoulder and pelvic girdles, and provides a large volume of its own movements in different planes. The muscles of the back, neck, shoulder, chest and hip determine the mobility of the vertebrae and the entire spinal column.

The spine is the basis of the axial skeleton and consists of five sections: cervical (7 vertebrae), thoracic (13 (12-14 vertebrae)), lumbar (7 (6-8 vertebrae)), and sacral. These 4 sections consist of 28 vertebrae, and between them there are 26 intervertebral discs. If we take into account 20 - 23 vertebrae of the caudal spine, their total number will be 50 - 53.

The cervical spine consists of 7 vertebrae. As a result of physiological lordosis, the connection of the cervical spine with the thoracic spine occurs at an angle of 30-40 degrees open dorsally, the intervertebral disc of the seventh cervical - first thoracic vertebrae (C7 - T1) is not visualised.

The body of the cervical vertebrae consists of two halves - the right and the left. The neck is the most mobile part of the spine. The vertebrae have considerable mobility relative to each other in a wide range of angles and amplitudes, so the dog can quickly turn its head in all directions by more than 180 degrees. Despite the various movements of the dog during jumping and sharp turns, the head always remains relatively stable, which is a mechanism for preserving the integrity of the brain from accidental injuries.

Cervical vertebrae 3-6 are the most similar in structure: they are large, have two branches of transverse rib processes and transverse foramina at their base. The articular processes are interconnected by lateral ridges, which results in a prism shape. Ventral ridges are present only at the caudal ends of the vertebral bodies. The spinous process on the third cervical vertebra (C3) is absent, and the length of the spinous processes on the others increases caudally. The rib processes are directed cranially. The dog cervical

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vertebrae are long, with a flat head and fossa, and the vertebral arches are wide. At the caudal end of the body of the axial vertebrae, the ventral ridge is clearly defined.

Atlantus (C1) - provides great mobility of the head. It has a ring-shaped structure with transverse processes in the form of wings. On the side of the spinal canal, the arch contains a surface for the tooth-like process of the axial vertebra. The lumbar processes have fused with the articular processes, making them very strong. In carnivores, the wings of the Atlantean are flat, thin, almost horizontal, and extended laterally. The wing pits are small. Instead of a wing opening, there is a wing notch. A transverse hole passes from the dorsal surface of the wing into the wing fossa.

The **axial vertebra** (C2) is the longest of all the vertebrae in the cervical spine. It is characterised by a large tooth-like process corresponding to the vertebral head, a spinous process in the form of a ridge, and unexpressed lumbar processes with lumbar foramina at the base. An intervertebral foramen opens in front of the lumbar process. The cranial articular processes are massive and are located behind and to the sides of the dentate process. In dogs, the latter is cylindrical, with the crest hanging cranially over the dentate process and merging caudally with the caudal articular processes. The intervertebral foramina are replaced by notches.

The seventh cervical vertebra (C7) has only one pair of rib surfaces at the caudal end of the body. The lumbar process is not branched, and there is no lumbar foramen at its base. The foramen is more robust than other vertebrae and is directed perpendicular to the vertebral body. In a dog, the vertebral head and fossa are flat, and the rib surfaces may be absent.

The thoracic spine consists of 13 vertebrae, sometimes there can be 12-14 vertebrae. The body of the thoracic vertebrae consists of four parts - two ventral and two dorsal. The thoracic spine is characterised by two pairs of surfaces for the rib heads on the vertebral bodies - in the head and in the fossa. The articular processes are not equally well defined on different vertebrae: from the withers, the processes gradually decrease in caudal direction.

The bodies of the thoracic vertebrae are cylindrical. From vertebrae 1 to 9, there is a gradual narrowing of their size, and then an increase in the size of the vertebrae in

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the caudal direction.

11 vertebra with a vertically standing spinous process is called an anticlinal **vertebra**. It has the shortest spinous process, which is triangular in shape. All the vertebrae behind it have spinous processes inclined cranially.

The thoracic vertebrae are rounded, with deep rib fossae on the first vertebrae, flatter on the following vertebrae, and absent on the last two or three. The spinous processes on the first 6-7 vertebrae are of equal length, and slightly thickened at the end. Their height and angle of inclination gradually decrease in the caudal direction. Articular, mastoid and accessory processes are clearly visible on the last vertebrae.

Lang and Loffler (1972**)** studied the cervical, thoracic and lumbar spine and found that the cervical spine is the most mobile in the vertical and horizontal planes. In the thoracic spine, in the area from the 1st to the 10th vertebrae, there is a possibility of movement. The amount of movement in the horizontal and vertical planes in the thoracic and lumbar spine is not the same everywhere. In the vertical plane, the range of motion decreases up to the anticlinal vertebrae and increases again behind it to the sacrum. In the horizontal plane, the maximum mobility occurs at the transition from the thoracic to the lumbar spine, and the minimum mobility occurs at the 5/6 vertebrae of the thoracic and lumbar spine.

The lumbar spine usually consists of 7 vertebrae. In dogs, there are often variations in the number of vertebrae: 6 or 8 vertebrae (6 vertebrae are common in dachshunds; according to Kusch (1983), one in seven animals). This phenomenon is called lumbalisation.

The surfaces of the articular processes are flat, with no ventral ridges. There are additional processes near the caudal articular processes. The lumbar vertebrae have the following features: flat heads and fossae; uniform development of spinous processes on all vertebrae, which are inclined cranially. The spinous processes are long. The mastoid processes are well developed and high. The body of the lumbar vertebrae consists of four parts - two ventral and two dorsal. In dogs, the lumbar vertebrae are long, with no ventral ridges. The spinous processes are long, flat and slightly narrowed. The lumbar processes are narrow and strongly deviated ventrocranially. Their length

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gradually increases to the 6th vertebra. The cranial articular processes have strongly pronounced mastoid processes.

The sacral spine is a monolith consisting of 3 sacral vertebrae that form the sacrum. Their bodies are decreasing in the caudal direction; they are separated from each other by lumbar lines. The spinous processes of the sacral vertebrae are fused into the middle sacral crest. The fusion of the sacral vertebrae into a single sacrum in different breeds takes place between six months and two years from birth. The body of the sacrum is curved (more so in females than in males), and the wings are turned in the sagittal plane. The cranial articular processes are almost of the same height as the spinous processes and have no breed-specific features. The boundaries of the spinal divisions are not stable: the last vertebra of a certain division can take on the morphological features of the neighbouring vertebra, imitating it. This process is called assimilation. It can be observed in all transitional boundaries of the spine. The orientation of the sacral vertebrae is straight.

The caudal spine consists of 20 to 23 caudal vertebrae. The first 5 to 6 vertebrae have arches and all the main parts. Their spines are awl-shaped and curved caudally. The cranial and caudal articular processes are distinct. The cranial articular processes have mastoid processes protruding from them. The lumbar processes are of considerable length, have a caudoventral direction and are thickened at the ends. From vertebrae 3 to 8, the caudal vertebrae have paired V-shaped hemal ossicles and are attached to the hemal processes through the medial joints, forming a bony hemal arch. In other vertebrae, the rudiments of the haemal processes at the anterior ends of the bodies form a characteristic thickening, which gradually decreases towards the last caudal vertebrae. The vertebral heads and fossae are flat-convex, and their bodies gradually shorten and thin in the caudal direction.

The spine has 4 curvatures: dorsally convex cervical, concave cervical-dorsal, slightly dorsally convex dorsal-lumbar and sacral. The sacral curvature in most cases is lordotic, but sometimes it can be kyphotic.

According to the anatomical classification, vertebrae are mixed bones. They have a complex shape and combine structural features of different types. Bones consist

of several parts that have different structures, contours and origins. Each of these parts has an inherent structure in accordance with the biomechanical functions it performs. The axial skeleton comprises the head, neck, trunk and tail.

When the vertebrae join to form the spine, the spinal canal is formed from the vertebral foramina, which is the container for the spinal cord. In a relaxed position with a uniform load on the thoracic and pelvic limbs, various intense movements of the spine are possible. All vertebrae are basically similar in structure, but at the same time they differ dramatically depending on their location and functions. The closer the vertebrae are to each other, the more similar they are, even if they belong to different sections. On the contrary, the further apart the vertebrae are, the more differences they have, even if they are in the same section. The body of each vertebra has vascular openings on its lateral and dorsal surfaces. Nerves leave the spinal cord and blood vessels enter it. For the passage of nerves and blood vessels, there are paired intervertebral foramina formed by the intervertebral notches of adjacent vertebrae.

Intervertebral disc. The elasticity and resilience of the spine, its mobility and ability to withstand heavy loads are determined by the influence of the intervertebral disc. It consists of two hyaline laminae, a fibrous ring and a pulp nucleus and is located between two adjacent surfaces of the vertebral bodies and is a complex anatomical structure.

In turn, it performs the following main functions:

1. The function of connecting and holding the vertebral bodies together.

2. A function of a semi-article that provides mobility of the body of one vertebra relative to another.

3. The function of the shock absorber, which protects the vertebral body from injuries and bruises.

The cranial and caudal surfaces of the 2 adjacent vertebral bodies are covered with cortical bone only in the peripheral regions, where it forms bony ring-like bands. In the centre, the surface is covered by a layer of dense spongiosis bone called the closing plate of the vertebral body.

The hyaline laminae fit snugly against the closing laminae of the bodies of

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adjacent vertebrae of the spine, which are equal in size. Cartilaginous hyaline plates are very dense and can withstand a fairly large load.

The fibrous ring consists of dense connective tissue bundles placed around the pulp nucleus and intertwined in different directions. The latter contains a small amount of intermediate substance and single cartilage and connective tissue cells.

The nucleus pulposus is a gelatinous mass consisting of a small number of cartilage and connective tissue cells and intertwined fibres.

An anatomical complex consisting of one intervertebral disc, two adjacent vertebrae with corresponding joints and ligaments at this level is called a vertebral segment.

The total length of all intervertebral discs, relative to the length of the spine (excluding the sacral region), is up to 16% in dogs.

The ventral longitudinal ligament, being a periosteum, is tightly fused to the vertebral bodies and passes freely over the disc. The dorsal longitudinal ligament, which participates in the formation of the ventral wall of the spinal canal, on the contrary, freely passes over the surface of the vertebral bodies and is fused to the disc.

In addition to the discs and ligaments, the vertebrae are connected by two intervertebral joints formed by articular processes that have features in different parts. These processes limit the intervertebral foramen, through which nerve roots exit. The arches and processes of adjacent vertebrae are connected by a system of ligaments: the yellow interosseous, supraosseous and intertransverse ligaments. The yellow ligaments are antagonists of the ligaments of the vertebral bodies; they functionally relieve the discs, preventing their excessive compression.

Nervous tissue makes up 2.0 - 2.5 % of the total body weight. It consists of nerve cells, nerve fibres, nerve endings and neuroglia. The structural and functional unit of nervous tissue is a neuron. Neurons are combined into organs, and organs into the nervous system, which perceives external stimuli [56].

The spinal cord is located in the spinal canal, which is formed by vertebrae and occupies 2/3 of its volume. Its absolute weight in a dog is 13 g, and its length depends on the length of the animal's body. In a medium-sized dog, the length of the spinal cord

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reaches 38 cm. The absolute weight of the brain in different breeds varies and ranges from 54 to 150 g. There is no clear anatomical boundary between the brain and the spinal cord, but the cranial edge of the atlas is considered to be a conditional boundary.

The dog's spinal cord is much shorter than that of other animals, ending at the level of 5 to 6 lumbar vertebrae and reaching a length of 45 to 70 cm. The relative weight is on average 0.12% of the body weight and is equal to 17 - 36 g. The spinal nerves are located in segments corresponding to a certain part of the spine and have their own names. From this it follows that the cervical spinal cord is designated (C1 - C7), the thoracic spine (T1 - T13), the lumbar spine (L1 - L7), the sacral spine (S1 - S3) and the caudal spine, caudal (Cd1 - Cd5).

The 7 cervical vertebrae form a movable but non-stretchable bone tube called the spinal canal, inside which the spinal cord is located and from which 8 pairs of spinal nerves extend. As a result of the large number of rotational movements of the cervical spine, the dog's spinal cord and spinal nerves do not feel compressed by the structures of the spine. The anatomical features of the spinal canal and intervertebral foramina in the cervical spine are much larger than in the thoracic and lumbar spine. The latter have limited mobility compared to the cervical spine. Due to the large lumen of the canals as a result of cervical trauma, compression pathologies with neurological phenomena are much less common. This is due to the fact that the spinal cord occupies less than half of its volume, while in the thoracic and lumbar spine, the spinal cord occupies almost the entire volume of the spinal canal. As a result of this "free" placement, compressive masses, such as haematomas, neoplasms, are accompanied only by pain syndrome, without causing significant compression of the spinal cord.

From a biomechanical point of view, the vertebrae of the cervical spine are subjected to predominantly compressive loads. As a result of walking, running or even jumping, the vertebrae and intervertebral discs experience significant compression loads that can damage their anatomical structure.

There are no pain receptors in the spinal cord, but they are present in the dura mater.

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1.2. Prevalence of spinal cord and spinal cord injuries in dogs

Intervertebral disc disease is most common in dogs of chondrodystrophic breeds (Shih Tzu, French Bulldog, Pug, Dachshund, Small Poodle, Basset Hound, Beagle, Pekingese). Statistically, this is 80% compared to dogs of the nonchondrodystrophic group, of which approximately 50% are dachshunds. The disease peaks at the age of 2 to 6 years. More than 50 % of all cases occur in the area of the twelfth to thirteenth thoracic vertebrae (T12 - T13) and the thirteenth thoracic to first lumbar vertebrae (T13 - L1) [83, 109, 113].

The localisation of intervertebral disc disease in the cervical spine ranges from 14 to 16 %, and from 84 to 86 % in the thoracolumbar spine [83, 109]. Accordingly, the presence of herniated discs in the cervical spine accounts for C2 - C3 vertebrae - 44 %, C3 - C4 - 23.5 %, C4 - C5 - 13.5 %, C5 - C6 - 13 %, C6 - C7 - 4.5 %, C7 - T1 - 1 %.

In the thoracolumbar spine, the statistical data indicate the following indicators: T10 - T11 - 0.9%, T11 - T12 - 11.5%, T12 - T13 - 26.5%, T13 - L1 - 25.4%, L1 - L2 - 12.7%, L2 - L3 - 8%, L3 - L4 - 7.3%, L4 - L5 - 5.5%, L5 - L6 - 1.5%, respectively.

Disc herniations in the thoracolumbar region are the most common cause of neurological disorders. Disc damage in this area accounts for 85% of clinical cases. Since disc herniations have been found in 84 breeds of dogs, chondrodystrophic breeds are in 1st place among other dog breeds. Subsequently, signs of UMN damage develop [83, 109].

Regardless of the breed of dog, intervertebral disc disease is diagnosed in an average of 0.25% of the total number of pathologies. Among dachshunds, this pathology occurs in one in four dogs [109].

In cases where an animal has undergone laminectomy without fenestration, recurrence can occur within 2 weeks with a probability of 18%.

The ascending syndrome is observed in 3-6% of dogs with serious neurological disorders associated with intervertebral disc disease in the thoracolumbar spine [110]. According to the latest data of Mankin J. M., the pathological process occurs only with

the development of type I intervertebral disc disease, often in the postoperative period within one to three weeks. The author describes the facts about the influence of bacteria before the onset of bacterial infections of the central nervous system. According to the data, they are noted in 2-5% of cases and are quite rare findings, but their role in the development of the syndrome has not been proven. The causes of development remain unknown, and the process cannot be modelled in clinical practice. With timely conservative treatment, up to 50 % of animals recover [49]. If 10 % of axons are preserved at the site of injury after spinal cord injury, this is sufficient for functional recovery and in 5 % of cases for the restoration of movement.

Myelographic examination: the statistical accuracy of the myelographic method is 85-95 %, and the incidence of complications is reduced to 1-3 %. According to the literature, the incidence of fatal outcomes due to complications of myelography is about 2 % [60-62].

Computed tomography: the sensitivity of CT in the diagnosis of Hansen type I intervertebral disc disease is 88.6 % (79.5 - 94.2), while the sensitivity of MRI is 98.5 % (94.1 - 99.7) [111, 112]. It was noted that CT is a less sensitive diagnostic method in dogs weighing less than 7 kg. According to the results of E. Kemelman's research, CT has 96 % accuracy in the diagnosis of type I and type II hernias in 48 animals out of 50 studied. In cases where the intervertebral disc is not mineralised, the diagnosis is significantly more difficult, as evidenced by studies in 48 dogs [112]. Based on the results of CT diagnostics in 23 dogs, the researchers obtained a 100% result in terms of the sensitivity of the method [111, 112]. With regard to the diagnosis of intervertebral disc disease according to Hansen type I, it has 99 % sensitivity to pathology and determines the degree of spinal cord compression relative to the volume of the nucleus pulposus in the spinal canal.

In the diagnosis of spondylosis, osteophyte formation was detected in 32 of 128 dogs (25 %). The interpretation of CT scans revealed 48 areas of spondylosis in these 32 dogs, 9 (18.75%) of which corresponded to intervertebral disc protrusion. To summarise the above data, the presence of osteophytes is not a reliable sign for the presence of IVDD-1 and its localisation in the intervertebral space [54, 55].

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1.3. Clinical forms of spinal cord and spinal cord injuries

To evaluate the autonomic nervous system and conduct a differential diagnosis of spinal cord and spinal cord injuries, the universal scale "Jaggy Etiology" is used, which is based on the acronym VITAMIN D:

V - vascular (cause), I - infectious, T - traumatic, A - abnormal, M - metabolic, I - idiopathic, N - neoplastic, D - degenerative. It can be used to determine the causative factors that cause neurological syndromes in dogs.

1.3.1. Cauda equina syndrome

Sauda equina, or "horse's tail", is a syndrome of damage to the caudal part of the spinal cord, which is a cluster of nerve roots that pass inside the spinal canal and exit the spinal cord segments at the level of the seventh lumbar - fifth caudal vertebrae (L7 - C5) [61].

During the prenatal period of osteogenesis, the dog's spine grows relatively faster in the longitudinal direction than the spinal cord itself. This causes the spinal cord segments to shift cranially in relation to the vertebrae. The spinal nerves extend from the respective spinal cord segments and pass caudally through the spinal canal to the intervertebral foramina. Accordingly, the sixth lumbar nerve leaves the SC within the fourth lumbar vertebra and passes caudally within the spinal canal to the intervertebral foramen between the sixth and seventh lumbar vertebrae. As a result, caudally from the third lumbar vertebra, the spinal canal contains a spinal cord of reduced diameter and nerve roots, which are called the cauda equina [44, 49, 61].

The lumbar spine in dogs consists of 7 vertebrae. They often have a deviation in the number of vertebrae: 6 or 8. In chondrodystrophic breeds, such as dachshunds, there are usually six vertebrae in every seventh animal.

The sacrum is formed by three fused vertebrae. Their bodies are decreasing in the caudal direction; they are separated from each other by the lumbar vertebrae and the intervertebral foramina.

The syndrome is most typically manifested as an association of symptoms at the

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level of the lumbosacral junction: peripheral paralysis or paresis of the distal pelvic limbs, dysuria (acute urinary retention or enuresis), and anaesthesia in the pelvic limbs and perineum. There is a characteristic severe radicular pain in the lumbar region. Conductive sensory disturbances often increase from top to bottom. In particular, there is a sharp decrease in body weight, muscle contracture, absence of achilles reflexes in the later stages, sensory impairment in the area of innervation of the damaged roots and vasomotor trophic disorders of the pelvic limbs.

The initial period of the disease is characterised by asymmetry of the symptoms of the injury. In some cases, Kernig's symptom and protective reflexes of the pelvic limbs are noted. The syndrome is often caused by injuries of the lumbar and sacral vertebrae, which are accompanied by haemorrhages in the sub- or epidural spaces. Sometimes, neoplasms in this area or a prolapsed IVD determine the clinical picture of the syndrome [112].

Nerve damage caused by nerve root compression can be exacerbated by ischaemia caused by blood vessel compression [73].

The horse tail syndrome occurs in dogs of different breeds, but in the vast majority of cases it is observed in medium and large animals.

Degenerative changes in the ACL, the so-called lumbosacral stenosis or lumbosacral syndrome, cause acute pain in the area of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1) in German Shepherds and their mixes, and in large breed dogs. Degenerative changes include proliferation of the intervertebral ligament, formation of osteophytes on the vertebral surfaces, degenerative intervertebral disc disease type II and deforming spondylosis in the area of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1). This leads to the development of spinal canal stenosis and compression of nerve roots in the area of the seventh lumbar vertebrae - the first sacral vertebra (L7 - S1) [61]. The syndrome is characterised by sharp radicular pain in the pelvic extremities. Over time, significant weight loss, contracture, sensory disturbances in the area of innervation of the affected roots, and vasomotor trophic disorders in the distal extremities are noted [113].

Diagnosis in some patients is difficult due to the variety of clinical signs and

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different etiological factors [111-113]. A thorough history, a complete clinical examination, and a detailed neurological examination can provide a preliminary diagnosis. Plain X-rays, in particular stress X-rays of the sacrum in flexed and dorsally extended positions, are considered valuable diagnostic steps to prevent instability in the area of the seventh lumbar vertebrae - the first sacral vertebra (L7- S1). RTG images do not show the degree of nerve compression, and sometimes such changes occur in dogs incidentally in the absence of associated symptoms [73].

Prognosis. In complicated cases, without surgical decompression of the spinal cord, no improvement in the general condition should be expected. A favourable prognosis is typical for patients with pain, but with a slight degree of neurological deficit. In general, the prognosis is from cautious to favourable for patients with moderate pain, moderate paresis and preservation of conscious control over the act of urination, if prompt surgical treatment is performed and an appropriate postoperative period of inactivity is provided. The prognosis is cautious or unfavourable for dogs with chronic back pain, prolonged pelvic paresis, and unfavourable for long-term urinary and faecal incontinence [73].

The pathophysiological process in the spinal canal is characteristic of spinal cord compression. Depending on the duration of the injury, tissue changes range from disseminated fibre degeneration to diffuse nerve fibre loss and fibrosis [113].

1.3.1.1. Caudal spinal cord injury syndrome

The syndrome was first described in 1954 by the German physician H. Verbiest, and in 1982 a detailed description was presented in the work of the neurologist M. D. Blagodatsky [113, 114]. The pathological process occurs when the cone is damaged and Cauda equina syndrome, the nerve roots of the lumbar spine, causes flaccid paralysis of the pelvic limbs in dogs and impaired urination and defecation [33, 34]. The symptom complex is characterised by the following clinical signs: absence of motor disorders, which may manifest as plegia or paresis in the distal limbs, sometimes with preservation of reflexes; absence of anal reflex or reduction of its sensitivity by the type of hyposthesia; peripheral urinary disorders (true urinary incontinence). The

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cone syndrome is caused by lumbar injuries, which are often accompanied by excessive haemorrhages that put pressure on the cerebral cone, and are often complicated by arachnoiditis or lumbar spinal neoplasms [115].

Epicondylar syndrome can be observed in case of simultaneous damage to the roots at the level of the fifth lumbar vertebrae - the first sacral vertebra (L5 - S1) and the cerebral cone, which sometimes manifests itself in acute cerebrovascular accident. In the event of a sudden onset of gait disturbance in a dog, it should be differentiated from caudogenic intermittent lameness syndrome, which manifests itself in the distal limbs and perineum, is acute in nature and is accompanied by flaccid muscle paresis and temporary absence of Achilles reflexes, which tend to regress within one hour. The syndrome is caused by circulatory disorders in the cauda equina with secondary hypoxia of the roots arising from the Cauda equina. The most common etiological factor is peripheral nerve disease at the level of the lumbar spine: lumbosacral pain and correlation deficit, hyporeflexia and paresis in the pelvic limbs, in particular the tail and anal sphincter, decreased perineal reflex, self-injury to the tail and pelvic limbs.

1.3.1.2. Lumbosacral syndrome

Degenerative changes in the lumbosacral spine, or the so-called lumbosacral syndrome (stenosis), cause acute pain in the area of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1) in German Shepherds and their mixes, in large and very large breeds. The degenerative changes include proliferation of the intercondylar ligament, formation of osteophytes on the articular surfaces of the vertebrae, degenerative disease of type II DM and deforming spondylosis in the area of the seventh lumbar vertebra - the first sacral vertebra (L7 - S1). As a result, it leads to the development of spinal canal stenosis and compression of nerve roots at the level of the seventh lumbar - fifth caudal vertebrae (L7 - C5) [61].

Vertebral malformations are most commonly found in the thoracic and lumbar spine in dogs with a brachiocephalic skull type, which include Boston Terriers, Pugs, English and French Bulldogs. The list of vertebral malformations includes: congenital malformation of half a vertebra, split vertebrae in the form of a butterfly, transitional

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vertebrae and vertebral block.

The above pathologies are incidental findings on examination radiographs, but sometimes these malformations cause spinal scoliosis and spinal cord compression. Ataxia and paraparesis can become more severe as the animal grows as a result of increased compression or stretching of the spinal cord (distortion due to downward pulling of the cauda equina). These injuries are manifested by excessive pain. Although vertebral malformations are visualised on conventional RTG images, spinal cord compression can be diagnosed using contrast myelography, CT and MRI scans. It is also possible to decompress the CSF, but this may increase the development of paraparesis or paraplegia to some extent. Some dogs with chronic compression may also have syringomyelia, hydromyelia, or spinal cord atrophy, which complicates conservative treatment and postoperative recovery time [61].

Degenerative diseases are characterised by the progressive degeneration of certain cell types in the nervous system. Such patterns include selective changes in neurons, myelin and axons with gliosis (hypercellularity), pallor or loss of white matter, spongy condition, etc. The vast majority of changes are observed in young animals of certain breeds with their slow progression. Some changes are confined to the CNS, but in lysosomal diseases, the accumulation also selectively involves external tissues. This selectivity leads to local neurological signs without signs of lateralisation. At the same time, there are no changes in the spinal cord [113].

1.3.1.3. Root syndrome

The radicular syndrome, which occurs as a result of nerve compression in the area of the sixth cervical - second thoracic vertebrae (C6 - T2) or the fourth lumbar - second sacral vertebrae (L4 - S2) with lateral displacement of the disc, is characterised by severe pain at the lumbar level [61]. The bulging of the intervertebral disc with the formation of a herniation can occur on one side of the dorsal longitudinal ligament that forms the bottom of the spinal canal. As a result, one of the limbs may develop more damage than the other. As a result, irritation of nerve endings is manifested by impaired support on the affected limb in a standing position and significant pain, which causes

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lameness during movement with possible development of muscle atrophy. In cervical discopathies, "radicular signs" may be observed due to atlanto-axial instability and soft tissue hypertrophy with the formation of stenosis in the intervertebral foramina. Clinical signs are characterised by claudication of the thoracic limbs, which occurs due to nerve compression and mild fibre loss until complete nerve root degeneration and fibrosis develop [113].

Timely diagnosis and qualified surgical care should prevent irreversible changes in the nervous tissue and restore motor function of the limbs in a relatively short time after spinal cord decompression. The purpose of the surgery is to remove fragments of the extruded disc and thus eliminate its excessive compression. Usually, the operation is performed for disc disease in the area of the tenth thoracic to fourth lumbar vertebrae (T10 - L4); lateral laminectomy is performed for disc herniation caudal to the fourth lumbar vertebra (L4), as the iliac bones prevent dorso-lateral access [61]. Decompression surgery for acute paraplegia and paresis is effective only in the first day after the onset of symptoms [61]. Limb function in dogs is restored within 48 hours only in 30-50 % of cases.

1.3.2. Schiff-Sherrington syndrome

Schiff-Sherrington syndrome is a rather life-threatening condition clinically manifested by increased extensor muscle tone in the thoracic limbs and paraplegia due to acute injury in the area of the third thoracic - third lumbar vertebrae (T3 - L3) [61]. The neurological syndrome is predominantly observed in severe SC injuries in the thoracolumbar region. In this case, spinal reflexes are correct, and sensation of the thoracic extremities and voluntary motor function are normal [30, 113].

In contrast to the thoracic spine, which is relatively stable anatomically, the strength of the connection between the thoracic and lumbar spine is much lower and the range of motion in different directions is much greater. This contributes to more frequent injuries to this segment of the spine, the degree of damage in injuries is much higher and the stability of the spine in injuries to the thoracolumbar region is often impaired. The centre of the posterior column of the spine can often undergo distortion,

while during rotation and flexion in the thoracic spine, the surface of the articular processes takes up a part of the energy of the interaction force [84].

The lumbar spine is characterised by a greater degree of free forward and backward flexion, as well as a certain degree of lateral mobility. Excessive rotational interactions result in tearing of the fibrous ring, ligamentous apparatus, and fractures of the articular processes, and excessive extension and flexion primarily affect the vertebral bodies [113].

According to Houlton and Taylor, Schiff-Sherrington syndrome is characterised by numb hypertensive thoracic limbs and weak hypotensive pelvic limbs [113-116]. Damage to the thoracic spinal cord affects inhibitory neurons, whose bodies are located in the cranial part of the lumbar spinal cord, and their axons project cranially, suppressing the function of the extensor muscles of the thoracic limbs [33]. The syndrome should be differentiated from seizures that occur in the event of damage to the SM in areas located cranial to the fifth cervical vertebra (C5) [34].

During the examination of the animal, muscle atrophy may be noted, which indicates the spread of the pathological process to the descending conductive pathways. If there is a serious damage to the SM of the thoracic spine with simultaneous injury to the ascending inhibitory pathways from the pelvic limbs to the thoracic limbs, the muscle tone of the extensor digitorum muscle may be increased. As a rule, this indicates a serious damage to the SM, which tends to form a rather cautious or unfavourable prognosis. From the point of view of pathological physiology, spinal cord concussion contributes to the development of spinal shock [79].

Spinal gait is manifested by impaired flexion and extension of the pelvic limbs due to increased reflexes 1 to 2 months after the development of severe damage in the area of the third thoracic - third lumbar vertebrae (T3 - L3). Using the functioning peripheral muscles, the animal can get up and try to move; these are involuntary movements, but they can be mistaken for voluntary ones [60]. Literature data indicate that the syndrome can occur when the spine is torn between the second thoracic to the fourth lumbar vertebrae (T2 - L4).

The onset of neurological symptoms is the result of direct spinal cord injury or

the progression of pathological changes in the spinal cord that develops later as a result of pathological mobility of the injured vertebrae. The main mechanisms of spinal cord injury are mechanical trauma, intervertebral disc disease, and displacement of bone fragments, which lead to narrowing of the spinal canal [113,114].

Depending on the degree of spinal cord injury, there are isolated spinal fractures in which victims do not show changes in neurological status, and complicated fractures with neurological disorders [83, 113].

Complete rupture of the SC in closed spinal fractures is quite rare. At the same time, preservation of the anatomical integrity of the latter may result in impairment of its function [84]. Spinal cord dislocation is caused by injuries that are accompanied by dislocation of more than 1/3 of the vertebral body. Acute cerebral oedema with insufficient blood supply occurs both as a result of trauma and as a result of the progression of oedema. As a result, impaired vascular innervation leads to stasis, haemorrhage, and, in case of prolonged blood stasis, necrosis of nervous tissue.

Schiff-Sherrington syndrome can be diagnosed 24 to 48 hours after a spinal cord injury. At the initial stage of diagnosis, it can be misdiagnosed as a result of the patient's general condition improving due to a careless examination. In such cases with severe injuries of the thoracic spinal cord, when several lobes of the brain may be involved, paralysis of the corresponding intercostal muscles may develop. Paradoxical breathing is noted, resulting in the coincidence of rib skeletons during inspiration and expansion during exhalation [44].

The main method of instrumental diagnostics of the nature and extent of vertebral damage in the acute period of spinal trauma is radiography. It helps to collect information about the condition of the spine and spinal cord in a short period of time. To determine the level of damage to the spine, preference is given to radiographs taken in the lateral projection.

While X-rays and CT scans show changes in the vertebral bone tissue, MRI can easily visualise damage to the ligamentous apparatus and intervertebral discs. MRI studies can additionally confirm a spinal cord tear [84].

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1.3.3. Intervertebral disc disease according to Hansen type I and II

The first diagnosis of intervertebral disc disease in dogs was recorded in 1881. It was described by Johnson in a dachshund dog.

Intervertebral disc diseases have been known since the nineteenth century, but their classification first appeared in 1952, proposed by Dr H. J. Hansen. This classification by the author is already considered relatively outdated after almost 70 years of use, but it is still used by most practitioners of veterinary medicine around the world.

Based on numerous MRI data, Turkish doctor Besalti O. identified 4 types of various degenerative changes in the intervertebral discs of the thoracolumbar spine [113, 114]: 1) disc degeneration; 2) bulging of the intervertebral disc; 3) disc protrusion; 4) disc extrusion. In veterinary medicine of dogs and cats, there is no similar approved unified classification of degenerative diseases of the IVD, which has led to great confusion with the terminology used [41].

According to the latest professional publications in the field of veterinary neurology, a new classification scheme for intervertebral disc disease has been developed. From now on, the scheme includes 7 types of hernias based on MRI examination of spinal cord and spinal cord injuries with simultaneous division into the period of the disease course. The high quality of the MRI scans made it possible to clearly visualise the integrity or damage to the fibrous ring with subsequent leakage of the pulp nucleus and the topical localisation of the intervertebral disc herniation. The results of the study are presented in Table 1.1 [113, 117].

Since 2015, a new type of intervertebral disc disease has been diagnosed using MRI diagnostics: hydrated disc extrusion. In fact, disc extrusion is a consequence of a previous protrusion with a sharp rupture of the fibrous ring and leakage of the pulp nucleus and refers to an acute course of the process.

It follows that several schemes for the diagnosis of intervertebral disc diseases in dogs are used in surgical practice, so a single and universal scheme has not yet been proposed. Accordingly, the total number of discogenic compressions has been constantly increasing since the discovery of the pathological process, and the

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development of new classifications requires a rather thorough and thorough work with sick animals.

Table 1.1 - Modern classification of intervertebral disc diseases in dogs (7 types)

Type of intervertebral disc disease	The course of the disease	Changing the disc
Hansen type I	Sharp	Extrusion of the calcified disc nucleus
Hansen type II	Chronic	Protrusion (fragments of the nucleus may be in the protruding ring)
Traumatic disc rupture	Sharp	A normal disc is compressed due to an external injury. The degree of compression depends on the condition of the disc
Non-compressive nuclear extrusions	Sharp	Normal hydrated disc
Acute annular extrusions	Sharp	Large fragments of the nucleus and ring are found in the lumen of the spinal canal. May be associated with massive epidural haemorrhage
Acute in chronic type I	Sharp	Large mass of calcified material with additional acute extrusion and spinal cord contusion
Acute in chronic type II	Acute, previous episodes are possible	Core protrusion and possible spinal cord contusion injury

Disc diseases are most commonly observed in dogs of chondrodystrophic breeds. Note that in veterinary surgery and neurology, there is no such disease as "discopathy". First of all, it is a generalised name for a group of intervertebral disc diseases that differ in their classification types and causal factors of their occurrence. On the other hand, it is a grouping of various disc-related diseases into one category, but it is not a mono-disease.

Gradual CBM bulging may not be clinically apparent until a certain point, as nerve tissue is able to withstand gradually increasing compression much better than

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rapid compression according to Hansen type I [113, 118].

It should be noted that degenerative diseases of type I and type II IVD can be observed in both chondrodystrophic and non-chondrodystrophic dog breeds. Occasionally, clinical cases of intervertebral disc disease in large breeds are observed in type I, and in chondrodystrophic breeds of dogs in type II. That is, there is no clear correlation that chondrodystrophic dog breeds suffer exclusively from type I hernia, and non-chondrodystrophic breeds - from type II, respectively. It is relatively rare, but possible for both types of degenerative disc disease to occur simultaneously in one dog.

The term "disc herniation" is incorrect and is not often used in modern veterinary vertebrology, as it does not reflect the essence of the pathological process, but is only a primitive simplification of the complex pathogenetic mechanism of the disease [41]. The bulging of the nucleus pulposus into the spinal canal can have the following locations in the segmental plane: median (central), subarticular, foraminal, and extraforaminal. Location in the sagittal plane: discal, infrapedicular, suprapedicular and pedicular, and "crescent".

Hansen type I intervertebral disc disease (acute course). It is characterised by the rupture of the fibrous ring with the spread of the pulp nucleus inside the spinal canal. In 1962, the scientist Funkquist described 3 types of disc jelly spread:

1. A pea-shaped spread of disc jelly is located only above the disc space and is classified as type I.

2. Spread along the body of the adjacent vertebra and is classified as type II.

3. Spread along the bodies of several vertebrae and is classified as type III.

In this case, the damage may remain localised and only a few segments of the spinal cord are affected, otherwise it may progress cranially and caudally and lead to autodestruction [230]. The pain syndrome can last up to 6-8 weeks, although more often it is noted up to 2 weeks.

Hansen type II intervertebral disc disease (chronic course). In the first 1.5 to 2 years of life, chondroid changes can already be observed in dogs with PD. With disc degeneration, dehydration occurs, and the nucleus pulposus is replaced by hyaline cartilage. The hydrostatic properties of the nucleus pulposus decrease, and the fibres of

the fibrous ring weaken. In dachshunds under 2 years of age, most of the discs undergo chondroid changes and their nuclei mineralise, usually changing their consistency from jelly-like to granular. With daily physical activity, the IVDs weaken, especially in the thoracolumbar spine. As a result, a large number of intervertebral disc diseases are observed between 3 and 6 years of age in most chondrodystrophic breeds. Hansen H. J. identified 2 main types of IVD degeneration: fibrous and cartilaginous metaplasia. In veterinary practice, this is a degenerative disease of IVD according to Hansen type I and II [41] or "intervertebral disc displacement".

Fibrous degeneration of the intervertebral discs occurs in NHP dogs after eight years of age and older as a normal aging process and is clinically evident after 10 years of age. The nucleus pulposus is also dehydrated, but it is replaced by fibrocartilaginous tissue. This occurs later than in chondroid degeneration, and the intervertebral discs are usually normal when the dog is young and active. The nucleus pulposus is not mineralised as often as in discs with chondroid or fibrous metaplasia. The disc itself deforms the spinal canal, causing myelopathy in adult dogs, which is called "intervertebral disc disease" [113, 119] or leads to protrusion of the fibrous mass through the partially torn dorsal part of the fibrous ring [32, 63]. The described type of hernia is most common in adult dogs of nonchondrodystrophic breeds, such as Dobermans, Rottweilers, and German Shepherds. The disease has a chronic course, with neurological deficits increasing gradually. Diseases of intervertebral discs can accompany diseases of the thoracolumbar spine in the form of discospondylitis, neoplasms at the level of the fifth lumbar vertebrae - the first sacral vertebra (L5 - S1), traumas and bruises. Over time, in some cases, spondyloarthrosis is diagnosed as a complication. Fibrous degeneration is rarely accompanied by mineralisation [113, 120]. Degenerative changes in the disc begin at the periphery of the nucleus, then move to the central part of the nucleus and the fibrous ring is involved. In dogs of nonchondrodystrophoid breeds, the pulp nucleus is gradually replaced by collagenous tissue.

A disc bulge with herniation can occur on one side of the dorsal longitudinal ligament that forms the bottom of the spinal canal. As a result, one of the limbs is more

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affected than the other. In case of nerve ending irritation, it is manifested by impaired support on the affected limb in a standing position and significant pain, which causes lameness.

The degenerative process in the intervertebral disc begins with the loss of proteoglycans with the transition of type II collagen to type I collagen, followed by proliferation of chondrocyte-like cells.

The cadaveric material contains a fairly large amount of extra-dural matter, which is often attached to the dura due to excessive bleeding. Significant disc protrusions often cause spinal cord contusion with haemorrhage, edema and diffuse necrosis of grey and white matter. In the chronic course of intervertebral disc disease, the nucleus pulposus material is organised by granulomatous inflammation and fibrous tissue that is tightly attached to the disc material in the dura [113].

1.3.4. Ascending syndrome

The brain and spinal cord are soft, friable structures that are particularly susceptible to external trauma. They are supported and protected in part by the dura mater and cerebrospinal fluid, and in part by the skull bone and vertebrae [121].

Traumatic injuries of the spine most often occur in the most functionally mobile parts of the spine: the cervical-thoracic, thoracolumbar and lumbosacral junctions [113, 121].

The main etiological factors that cause the development of degenerative myelopathy are classified according to the following criteria. 1. traumatic nature (fracture, luxation, subluxation of vertebrae). Diseases of intervertebral discs according to Hansen type I (clinical signs are acute and non-progressive). 2. Vascular pathologies: embolism of the fibrocartilaginous ring (acute and painless) [49, 113].

One of the causes of spinal cord compression may be Hansen type II intervertebral disc disease. It should be noted that degenerative intervertebral disc disease of various types can be observed in most breeds of dogs. In particular, the simultaneous presence of both types of degenerative disc disease is also possible [97, 133]. When a herniation occurs, compression and traumatic concussion of the spinal

cord develop, resulting in neurological signs. Contusion leads to axonal rupture and demyelination of nerve fibres, haemorrhagic necrosis of the grey matter of the spinal cord and decreased blood flow. The metabolic mechanism is caused by ischaemia as a result of the primary injury and leads to an increase in the formation of free radicals that exceed the capabilities of the body's defences. The vascular mechanism causes further development of spinal cord ischaemia caused by primary and secondary metabolic processes. The spinal cord is able to withstand compression and compensatory loads for a certain period of time, so for ease of diagnosis, clinical signs were grouped into 6 categories. Accordingly, category V is associated with spinal cord compression ischaemia. If the latter is not eliminated within 24 to 36 hours, degenerative myelomalacia gradually develops as a result of circulatory disorders and edema, which can cause destructive changes in the spinal cord substance. According to statistical data, these include 10% of injuries to the thoracolumbar spine. Category VI includes patients with ascending myelomalacia that has already developed after spinal cord injury or has recently developed. In this case, abduction and adduction of the pelvic limbs are impaired, and clinically observed paraplegia progresses to tetraplegia [62].

Ascending myelomalacia is the destruction of spinal cord tissue by its softening or lysis, destroying the parenchyma and roots of the spinal nerves that pass to the diaphragm and intercostal muscles. The herniated nucleus pulposus of the intervertebral disc due to the rupture of the fibrous ring causes rapidly developing ischaemia of the spinal cord tissue, which is associated with irreversible damage to the spinal cord tissue [113]. As a result, the respiratory system dysfunction occurs, leading to asphyxiation and death of the animal. Progressive myelomalacia develops in a few days after the onset of limb paralysis. In this case, severe depression, hyperesthesia, progressive loss of pelvic limb reflexes are clinically noted, and the response area in the pannicular reflex test is shifted cranially. There is literature evidence that ascending syndrome may be associated with genetic disorders of the superoxide dismutase (SOD1) enzyme in certain dog breeds, such as the German Boxer and Pembroke Welsh Corgi [122].

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Clinical diagnostic methods are based on a clinical and neurological examination, which should result in a neuroanatomical diagnosis describing the most likely localisation of the pathology, in accordance with the division of the spinal cord into the first cervical - fifth cervical vertebrae (C1 - C5) and the sixth cervical - second thoracic vertebrae (C6 - T2). **In** particular, digital radiography does not identify the spinal cord, so it is not possible to assess the degree of compression and identify the area of the damaged spinal cord segment. Therefore, the prognosis for lower motor neurons is often poor, and for upper motor neurons, the prognosis is generally cautious. To establish a definitive diagnosis, it is recommended to use a magnetic resonance imaging scanner, as it is impossible to visualise the spinal cord by X-ray without the use of contrast agents. Given the risk of developing ascending syndrome after spinal surgery, the use of MRI diagnostics is advisable in practice [123]. MR imaging better shows soft tissues, such as muscles, cartilage, blood vessels, and spinal cord elements [62].

If animals survive with ascending syndrome, the pathological process may stop at the level of one compartment. Accordingly, there is no recovery of tactile and pain sensitivity, but spontaneous recovery is rarely observed. Limb function and movement are impossible. No preventive measures have been developed to prevent the development of the syndrome due to unknown etiological factors [124].

The use of a contrast agent in myelography allows detecting changes in meningeal structures when the blood-brain barrier becomes permeable. In this case, some spinal cord injuries can be diagnosed in a timely manner. The reason for poor visualisation of the level of the damaged intervertebral disc on myelograms is spinal cord edema, which blocks the contrast column over several vertebrae and several levels of intervertebral discs, respectively. As a result, there are grounds to characterise myelography as a sensitive method for determining the location of spinal cord compression, but it does not allow determining the etiological factors that caused the injury.

Normally, the contrast medium around the spinal cord appears on radiographs as two parallel bands, a ventral and a dorsal column. Without contrast medium, the

contour of the spinal cord is not defined, only the contours of the vertebral bone tissue are visualised. In the case of spinal cord contusion in the first hours after the injury, myelography is performed to identify the area of spinal cord edema. If myelomalacia develops, the contrast agent can mix with particles of necrotic tissue in the damaged area of the spinal cord, which also leads to the appearance of turbidity or heterogeneity in the fluid. If the disease is not acute, myelography indicates local spinal cord atrophy.

At autopsy, the spinal cord is liquid, jelly-like, and can even be drawn into a syringe if necessary. Malation is defined as microscopic softening and necrosis of the tissue as a result of the destruction of most cells in the affected area, which leads to a complete loss of the primary structure. Complete destruction by cavitation is easily detected, whereas acute lesions are characterised by a jelly-like consistency, often with a yellow or grey discolouration. Histologically, malignant lesions are separated from normal tissue and are paler or even darker than the surrounding tissue. The primary structure is lost, and the affected tissue appears less compact, vacuolated, and with widespread cell necrosis [113, 124].

1.4. Examination methods for spinal cord and spinal cord injuries

1.4.1 Orthopaedic examination

Before conducting a neurological examination, there is an urgent need to differentiate between diseases of the musculoskeletal system in dogs that may be similar in clinical signs to neurological diseases. The scheme of orthopaedic examination includes the following main tasks:

1. Determine the presence or absence of lameness.
2. Evaluate the orthopaedic or neurological nature of the lameness.
3. Determine which limb is affected.
4. Determine the degree and nature of the lameness.
5. Determine the location of the pain.
6. Conduct special orthopaedic tests.
7. Formulate differential diagnoses for the identified diseases of the

musculoskeletal system.

8. Perform instrumental and laboratory tests to confirm the diagnosis.

9. Determine the further tactics of assistance.

10. Formulate a prognosis in the case of a particular method of treatment (conservative or surgical), as well as in the absence of it.

As a result of the examination, we establish the presence of pain syndrome, which can be caused by such factors as:

1. Lameness as a result of "orthopaedic" pain due to injury is an injury to the joint capsule, bones, muscles, tendons and ligaments.

2. Lameness as a result of "neurological" pain - occurs when the nerve root is compressed and damaged.

3. Mechanical lameness "without pain" - joint arthrosis, contractures, chronic joint dislocations, severe limb deformities (Table 3.2 in Chapter 3).

The main element in the study is a detailed medical history, where data on the animal is recorded, from breed, age and sex to the treatment performed in veterinary medicine centres. After this stage of work, the dog is monitored from a distance for abnormal posture of the thoracic and pelvic limbs associated with genetic diseases, dislocations, subluxations, valgus and varus bone deformities, developmental abnormalities, etc. The next stage of the examination is a clinical examination and palpation of the limbs for changes in the total muscle mass, anatomical malformations, swelling or thickening of the limbs in the joints, the presence of pathological mobility, and the determination of pain.

According to the examination scheme, the thoracic limb is first worked on, where special orthopaedic tests are performed: forearm supination/pronation test; compression test in the area of biceps tendon attachment; compression test in the area of the hook-shaped process; biceps stretching test; full flexion + shoulder abduction/adduction; full extension of the shoulder + compression in the caudal neck area.

Examination of the pelvic limb includes assessment of the knee joint under sedation: retractable shuffle test; Henderson compression test; assessment of

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pathological mobility of the hip joints; Ortholani test at the first stage with subluxation of the head (Barlow test, equal to 33 degrees) and at the second stage of head repositioning (Ortholani angle, equal to 58 degrees). The examinations are performed using a medical goniometer. Accordingly, after conducting a comprehensive examination of the musculoskeletal system for the presence of bone or muscle and tendon damage, the data obtained are summarised and decisions are made regarding further diagnostic manipulations.

1.4.2. Neurological examination

Neurological examination is a rather complicated, yet reliable way to assess the functional state of the spinal cord's conduction pathways and segments. They help determine the location, severity of spinal cord injury and the probable cause of the disease.

To determine the neurological status of a sick animal, various diagnostic methods are used, which often unreliably characterise the patient's condition at the time of clinical examination, therefore, in order to unify the results obtained in human medicine, a single classification of neurological examination was proposed with the sole purpose of obtaining an objective assessment (Table 3.3).

The International Standard for the Neurological Classification of Spinal Cord Injury was first developed by the American Spinal Injury Association (ASIA) in 1982 to unify data from the National Spinal Cord Injury Statistics Centre. In 1989 - 1990, the ASIA committee identified key muscle groups and sensory points to determine the extent and level of spinal cord injury [124].

The most significant changes to the guidelines were made in 1992, 1996 and 2000. The second edition of the practical guide was published in 2003.

Key muscle groups are examined from rostral to caudal segments. ASIA recommends that the level of impairment be determined by the lowest segment with preserved function [113, 124]. The ASIA committee defined complete damage as the absence of sensory and motor function in the anus and perineum, which are innervated by the corresponding spinal cord segments [79].

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1.4.3. Examination of cerebrospinal fluid

Cerebrospinal fluid is a fluid produced by the vascular plexuses of the ventricles of the brain. It circulates through the ventricles of the brain and is reabsorbed through the arachnoid membrane. The composition of CSF can change as a result of pathology affecting the outer surfaces of the brain and spinal cord [65].

On the other hand, cerebrospinal fluid is a clear, colourless ultrafiltrate of blood plasma that fills the ventricles and subarachnoid space of the brain and spinal cord. Surrounding the brain substance, the fluid acts as a buffer, which reduces the possibility of injury. It performs immune and trophic functions, ensuring the constancy of the internal environment of the CNS regardless of fluctuations in the blood composition and removes metabolic products of nerve cells [64, 65, 92].

This is the only easily accessible tissue that allows for an objective assessment of the state of the central nervous system. The examination is recommended even in the absence of abnormal haematological parameters, in particular, in general and biochemical blood tests. Cerebrospinal fluid sampling is performed immediately before contrast myelography (if necessary). Its repeated examination determines the effectiveness of the treatment, and also makes it possible to obtain baseline data before discontinuing treatment. For the analysis, 1 ml of fluid is taken at a flow rate of 1 drop/second or slower. It is advisable to use a red-cap tube rather than an EDTA tube, as CSF does not contain clotting factors, and EDTA causes an increase in the measured protein concentration, while diluting the sample, which itself contains a low concentration of cells.

For reliable results, cerebrospinal fluid should be examined within 30 to 50 minutes of collection. Recent publications indicate that the total cellularity does not change after 24 to 48 hours of sample storage, but the percentage of cells changes significantly, which in turn can misinterpret the data. A hand-held haemocytometer or a Fuchs-Rosenthal chamber is used for the test. When performing the cytological diagnostic method, the sample is evaluated semi-quantitatively, even if the amount of fluid is insignificant for other tests.

The results of the study should be interpreted in conjunction with clinical

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findings and diagnostic findings during X-ray or MRI examination. It should be noted that bacteria are not detected in CSF. They can be found only in 2 cases: when taking fluid from cadaveric material and when examining macrophages.

In case of spinal cord diseases, a puncture to obtain CSF is performed in the lumbar spine, and in case of brain diseases - in the occipital-Atlantic region.

1.4.4. Myelographic examination

Myelography is one of the radiological examination methods that provides visualisation of the spinal cord contours after injection of contrast agent into the subarachnoid space [62]. Myelography with the injection of a positive contrast agent lipiodol into the subarachnoid space of the spinal cord was first proposed by Sicard and Forestier in 1921 [48, 92]. Descriptions of myelography, its techniques, indications and contraindications for the use of this diagnostic manipulation are also published in the literature, so today the history of studying the issue of spinal cord and spinal cord injuries in dogs has more than five decades [54, 55]. Myelography began to be widely used in veterinary medicine after the popularisation of this technique by Dr Hoerlein in the late 50s [113, 125].

Normally, the contrast medium around the spinal cord is displayed on plain radiographs as two parallel bands, a ventral and a dorsal column, extending from the occipital process, if injected into the occipitoatlantal foramen, to the sacral vertebrae. Without contrast medium, the spinal cord contour is not visualised, except for the vertebral bone tissue [69]. This is explained by the occurrence of spinal cord edema, which blocks the contrast column over several vertebrae and levels of intervertebral discs, respectively. Thus, there are grounds to characterise myelography as a sensitive method for determining the location of spinal cord compression. The main advantage of the method is its ease of use, availability of contrast agents and high reliability in determining spinal cord compression.

When contrast myelography is used, poor filling of the subarachnoid space may be noted, indicating cerebral edema. The use of a contrast agent can detect changes in meningeal structures when the blood-brain barrier becomes permeable. In this case,

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myelography can also detect some spinal cord injuries with the determination of the degree of compression on radiographs [75]. The method also allows visualising the contours of the spinal cord and is effective in diagnosing Hansen type I intervertebral disc disease. Compared to tomographic methods, it remains the basic method for diagnosing spinal cord injuries.

Some peculiarities should be taken into account when performing RTG diagnostics. If there is no caudally elongated dura mater sac above the lumbosacral junction, the diagnostic capabilities of the myelogram are limited and it should be supplemented with modern cross-sectional images [73].

In case of acute course of the disease, myelography is performed to detect the area of spinal cord edema. If myelomalacia develops, the contrast agent may mix with particles of necrotic tissue in the damaged area of the spinal cord, which causes it to appear cloudy or heterogeneous. If the disease is chronic, myelography indicates normal or localised atrophy of the spinal cord [44].

In particular, there is a danger when performing myelography. Since the needle is inserted into the subarachnoid space, it can injure the nerves that extend from the spinal cord, shifting them in different directions. In the absence of spinal cord swelling, the likelihood of nerve damage is negligible. Most adverse reactions after myelography occur within a few hours after injection of the contrast agent as a result of slow absorption of the drug and its distribution mainly by diffusion throughout the body.

It should be noted that the more the patient moves or strains after injection of the contrast agent, the faster it mixes with biological fluids of other areas not related to the study site. As a result, the contrast density decreases. Drugs used for intravenous administration should not be used, as they are toxic to nervous tissue and their use can lead to severe neurological complications or death [32, 33, 69].

Complications of myelography. Sometimes seizures or neurological disorders, increased intracranial pressure may be detected. The use of Tomohexol reduces the risk of side effects. Serious neurological complications occur in case of violation of the diagnostic procedure technique. Severe complications of both the nervous and cardiovascular systems (ventricular arrhythmia) may be observed if a contrast agent is

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injected into the central canal during cisternal myelography. Lumbar myelography cranial to the area of the fifth lumbar - sixth lumbar vertebrae (L5 - L6) can cause lumbosacral edema, and therefore this procedure is not recommended [113, 124]. In particular, clonic seizures may occur when contrast medium is injected into the cistern, but they rarely occur with modern agents. Cases have been described in which iatrogenic meningitis can be caused by injection of contrast medium into the occipito-atlantic foramen [113].

Contrast medium may enter the central canal of the spinal cord iatrogenically. If the amount of injected substance is small, the likelihood of neurological dysfunction is an uncommon clinical sign.

According to the literature, the main contraindications for myelography are inflammation of the meningeal membranes in the form of meningitis, myelitis, and atlanto-axial instability [126]. The total amount of iodine in the CAP should not exceed 3 grams [53]. Tomohexol is excreted by the kidneys, so the administration of the substance is contraindicated in animals with renal insufficiency. Contrast agents contain iodine, so they should be used with caution in patients with hyperthyroidism [35].

Myelography is necessary to determine the site of compression in dogs with spinal cord injury. This diagnostic procedure may be indicated for use in cases where patients are presented for decompression surgery, where the exact level of injury to the spinal column remains unknown. Nevertheless, information obtained from plain radiographs in combination with clinical examination is usually sufficient [119].

1.4.5. X-ray examination

Since 1895, the year of the discovery of X-rays, X-rays have been the only method of displaying static diagnostic images. For medical radiography, electrons are accelerated in X-ray tubes with voltages of 120 to 150 kV. They bombard the anode of the tube and knock out X-ray quanta with energy from 10 to 150 MeV. In the twentieth century, analogue technology prevailed to produce still (radiography) and moving (fluoroscopic) images using various imaging systems on monitors.

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Digital radiography first became a reality in the late 1980s, when Dr Francis Mouyen created the RadioVisioGraphy (RVG) system.

International documents formulate the principle of dose formation in X-ray diagnostics - ALARA - As Low Reasonably Achievable - (as low as possible).

The main advantage of X-ray radiography is its accessibility, high diagnostic value, and non-invasiveness. This method is used to diagnose injuries to the spinal column, but does not allow for a proper assessment of the degree of spinal cord damage. As a result, at the first stage of examination, X-ray radiography is performed to detect visible changes in bone tissue, vertebrae, and signs of Atlanto-axial instability. The main changes diagnosed by X-ray radiography are defects in vertebral development, assessment of vertebral positioning relative to each other, and homogeneity of their structure. Less commonly, changes in intervertebral discs with their mineralisation are detected. If the data obtained are not sufficient to establish a diagnosis, it is recommended to perform an MRI or CT scan to visualise soft tissue structures.

The intervertebral discs, due to the physiological curvature of the thoracic spine axis and projection distortions, are visualised in all segments using the lateral or ventrodorsal projection (VD projection). The correct anatomical relationship is indicated by two lines drawn along the upper and lower contours of the vertebral bodies. The lines should be smooth and parallel to each other, in particular, slightly curved. An image of each pair of ribs is superimposed on the vertebral bodies of the thoracic spine, which may projectionally merge into one shadow. In the ventrodorsal view, the articular processes are connected to each other in a "tile" type. The size of the vertebral bodies gradually increases in the caudal direction up to the 6th lumbar vertebra, and the length of the seventh lumbar vertebra (L7) decreases.

Nerve roots, spinal cord, and intervertebral hernias are not visualised on radiographs. An exception to this is the increased radiological density of the intervertebral disc with pronounced mineralisation, which indicates dystrophic changes.

Obtaining high-quality X-rays requires the correct positioning of the animal's body on the X-ray table. The spinal column is placed parallel to the surface of the

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cassette of the appropriate size, soft pillows are placed under the sides, and plastic positioners can be used for the thoracolumbar region.

An examination X-ray is performed to exclude serious pathologies, such as spinal neoplasms, trauma, pathological fusion and changes in the shape of vertebrae, fractures of vertebral bodies, bones, etc. Digital radiography in neurology has limited diagnostic value, but it is necessary for differential diagnosis.

To prevent technical errors that could arise during the diagnostic process, dogs are administered drugs to ensure a state of severe sedation and anaesthesia (subsection 3.1.2).

1.4.6. Tomographic examinations

1.4.6.1. Computed tomography (CT)

On 8 November 1895, Wilhelm Conrad Roentgen discovered X-rays. Already in 1967, Jack C. Geary expressed the opinion that a computed tomography scanner was necessary for use in veterinary medicine. The first computed tomography scanner was opened in 1969 by Godfrey Newbold Hounsfield and Alan McLeod Cormack. In 1973, Christine Gibbs began researching linear tomography in veterinary medicine. The main focus for the scientists was the mention of the clinical value of obtaining layer-by-layer images for the diagnosis of various pathologies of the axial skeleton and spine [113, 127]. In 1979, Cormack and Hounsfield received the Nobel Prize in Physiology or Medicine. In early 1995, it became possible to perform real-time reconstruction of tomograms, and in the period from 2005 to 2007, the number of layers increased to 64. In 2008, Siemens introduced a new generation of scanners that can compile images in less than a second.

CT is a non-invasive diagnostic method based on X-rays. The CT method has maximum sensitivity to pneumatised and mineralised structures, in particular, CNS tissue. The main plane for the study is the segmental plane, which is used for the main diagnosis of CAT (Computed Axial Tomography).

To visualise the density of structures, a scale of attenuation of RTG radiation called the Hounsfield scale is used. This is a range of units of the scale of "densitometric

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indicators" called Hounsfield units, which correspond to the level of attenuation of X-rays by body structures, ranging on average from -1024 to +1024. The average value of the Hounsfield scale (0 HU) corresponds to the density of water (Table 1.2).

CT scanning involves a slice thickness of 1 to 3 mm, and on average, during the study period, we receive about 100 to 200 CT scans that can be interpreted for the presence of a pathological process.

Table 1.2 - Indicators of radiological density

Density of the pulp nucleus substance in the spinal canal*.	417.5 ± 14.81 HU
Average density of the spinal cord	126.3 ± 5.45 HU

** Rarely below 200 HU*

Kemelman E. conducted a study on the use of CT in the diagnosis of IVDD-1. As a result, the average density of 160 IVDD-1 in the spinal canal was 417.5±14.81 HU [54, 55]. The average density of the pulp nucleus in the usual anatomical location was 886.0±10.64 HU. This may be due to the fact that the mass determined on CT is not only the substance of the pulp nucleus, but also contains fragments of the fibrous ring and blood. In 23 % of dogs (22.1 %), osteophytes corresponded to IVDD-1, but not necessarily in the area of spinal cord compression. Of these, 58 dogs had neurological disorders, and 48 had various diseases [113, 128].

Quite rarely observed in dogs is the "vacuum phenomenon", which is characterised by the accumulation of free air in the disc tissue and released into the cavity by diffusion. It contains carbon dioxide, oxygen and nitrogen, which is poorly absorbed into the avascular tissue. The phenomenon is not considered a reliable sign of IVDD-1 and does not depend on the duration of the disease and the severity of neurological signs, but is more common in the presence of IVDD-2. Out of 160 dogs studied, the "Vacuum Phenomenon" was detected in 2 animals, which made up 1.25% of the total number. Summarising the data, the "vacuum phenomenon" is not a differential sign for IVDD-1 [54, 55].

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1.4.6.2. Magnetic resonance imaging (MRI)

One of the newest methods of radiation diagnostics is magnetic resonance imaging (MRI). The method is based on the phenomenon of nuclear magnetic resonance, which has been known since 1946, when F. Bloch and E. Purcell showed that individual nuclei located in a magnetic field induce an electromagnetic signal under the influence of radio frequency pulses. In 1952, the scientists were awarded the Nobel Prize for the discovery of magnetic resonance. In 2003, the Nobel Prize in Medicine was awarded to British scientist Peter Mansfield and American scientist Paul Lauterbur for their significant contribution to the development of MRI. In the early 70s, Paul Lauterbur discovered the possibility of obtaining 2D images by creating a gradient in a magnetic field. In the late 1960s, D. Hutchinson began to study MR and electron paramagnetic resonance in mice. In turn, Damadian R. measured the T1- and T2-relaxation times of normal and tumour tissue samples from rats.

In the mid-80s of the twentieth century, MRI scanners appeared in Germany. At that time, Dr M. Sager and Dr Assayer began working on an atlas of MRI anatomy of dogs. In 1997, the atlas was published and recommended for the study of MRI in animals. The tomograph is based on a magnet that generates a magnetic field, additional excitation coils and a signal receiver. Resistive magnets produce a relatively low magnetic field strength of 0.3 Tesla. The nature of the MR image is determined by three factors, such as proton density, T1 and T2 relaxation times. The latter produces images (T1) ranging from bright white in fluids and fat to black in bones. Therefore, the images obtained on the basis of T1 and T2 relaxation times are related to each other as negative to positive, which allows contrasting certain tissues.

MRI is the "gold standard" for diagnosing diseases of the central nervous system, spinal cord compression injuries, and brain damage. The method allows you to clearly visualise intervertebral discs, the degree of their protrusion and spinal cord swelling. The disadvantages of the method are the presence of metal implants after surgical interventions. The power of the MRI machine is also of great importance. Equipment with a power of 1.3 Tesla is recommended for use in veterinary medicine, although experience with 0.3 Tesla tomographs also makes it possible to obtain high-quality MR

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imaging. The examination time is 25-30 minutes, in other cases it can reach 40 minutes, depending on the location of the pathological process. Correct positioning of the animal provides reliable MR images in the segmental and sagittal planes.

MRI is a non-invasive diagnostic method. One of the main disadvantages of this method is that it takes 30 to 40 minutes to complete the examination, compared to CT, which takes only 1 to 5 minutes. In particular, MRI examination provides high reliability of the results obtained in soft tissues, such as the spinal cord, etc. As for the diagnosis of intervertebral disc disease according to Hansen type II, it is impossible to reliably determine the compression of a single disc protrusion.

In chondrodystrophic dog breeds, MRI is a more accurate and rapid method of diagnosing IVDD than contrast myelography [41, 53]. For a more accurate diagnosis, a multi-plane reconstruction can be performed when interpreting an MR image. The intervertebral discs are quite well visualised on MRI and have a "glowing" effect. This is due to the fact that the discs contain a lot of water and jelly-like substance, which has a luminescence effect in STIR T2 mode.

STIR mode (Short tau inversion recovery) is a sequence of STIR spin echo inversion, the so-called short T1 inversion recovery, is a method of signal suppression with an inversion time T1 = T2 In2 at which the signal from adipose tissue is zero. In a magnetic field of 1.5 T, this corresponds to approximately 140 ms. The mode described above is used to diagnose brain tumours and traumatic brain injuries.

FLAIR (Fluid attenuation inversion recovery) mode is a long T1 inversion-recovery sequence used to eliminate the influence of fluid in the image. It is used for CNS diseases: subarachnoid haemorrhage, spinal cord infarction, meningitis and traumatic brain injury in dogs.

1.5. Methods of treatment of spinal cord and spinal cord injuries

Timely diagnostics and skilfully performed surgical care help to prevent irreversible changes in the nervous tissue and restore the motor function of the limbs in a short time after spinal cord decompression.

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The purpose of the surgery is to remove fragments of the extruded disc and eliminate compression. The surgery is usually performed for disc disease in the area between the tenth thoracic vertebrae and the fourth lumbar vertebrae (T10 - L4); lateral laminectomy is performed for disc disease caudal to the fourth lumbar vertebra (L4), as the iliac bones prevent dorso-lateral access.

Before surgery, the animals are carefully prepared, the surgical field is prepared, in particular, the trachea is intubated and the patient is put into a state of anaesthesia by inhalation of an inhalation anaesthetic. A pulse oximeter is attached to the tongue. In case of bleeding, a bipolar electrocoagulator is used. The holes in the vertebrae are formed with a drill, and the remaining bone tissue is irrigated and aspirated with a surgical suction.

The technique of surgical intervention is described in Funkquist (1962), Schulman a. Lippincott (1987), Black (1988), Wheeler (1988).

Depending on the pathological process, there are 2 methods of treatment: surgical and conservative (caging for 14-21 days).

If, for some reason or contraindication, surgery to remove the extruded disc substance cannot be performed, the animal is kept in an enclosure without the use of analgesics. This is because with high-quality anaesthesia, the dog begins to move actively, which in turn can further injure the intervertebral discs and cause the substance to spread into the spinal canal.

Treatment of Hansen type I intervertebral disc disease is performed surgically using the Ventral Slot method. In this case, the access is made from the bottom of the vertebra, i.e. ventrally, and the jelly-like substance of the intervertebral disc is fenestrated through the formed window. The basic rule is that only the disc fragment that directly causes compression of the SM is removed. The main postoperative consequences after surgery are: a fluid-filled cyst is formed; long-term compression of the spinal cord by the previously extruded substance, even after surgery, can cause deformation of the latter.

Hemilaminectomy. The dog is fixed in a supine position so that the side of the body to be operated on is elevated. The skin is incised laterally from the dorsal midline

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of the body, starting from the end of the thoracic spine and continuing caudally along the spine. According to conventional methods, we cut the tissues and open access to the vertebrae. Then, using a 2-3 mm thick cutter, we widen this distance and open access to the lateral surface of the spinal cord. The notch in the bone is extended horizontally up to half the length of each of the adjacent vertebrae adjacent to the damaged disc. After the operation is completed, fragments of the displaced intervertebral disc are removed from the bottom of the spinal canal using an elevator or curette. The final removal of all intervertebral disc residues is performed by flushing the surgical site with 0.9% NaCl saline with a syringe and catheter. Before the final wound closure, fenestration is usually performed in adjacent intervertebral discs for prophylactic purposes.

The main indications are bladder paresis and intervertebral disc disease of type I and II. Technical difficulties: access to the contralateral location of the hernia is limited; ventral access to the disc is also limited if there is fusion with the vertebrae, which causes difficulty in its resection; instability may occur when performing the operation on both sides.

Mini-hemilaminectomy. The operation, which involves the removal of fragments of displaced discs from the spinal canal, can be performed together with lateral fusion of the intervertebral discs of the thoracolumbar spine [97, 109, 133]. Mini-hemilaminectomy is performed in the area immediately dorso-lateral to the damaged intervertebral disc. In this case, the intervention does not cover the tissues located above the adventitious process of the vertebra. As a result, the articular surfaces of the vertebrae remain intact. Fragments of the displaced intervertebral disc are removed from the bottom of the spinal canal as described above in the hemilaminectomy technique.

The method allows to achieve spinal cord decompression with minimal tissue trauma and avoid damage to the articular surfaces of the vertebrae. It is easy to perform simultaneously with fusion of adjacent vertebrae. If a mini-hemilaminectomy is not possible, an operative access can be created by enlarging the holes for several vertebrae and completing the manipulation as a conventional hemilaminectomy. The main

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indications are type I and type II DM disease.

Fenestration is a surgical procedure to remove the remnants of the pulp nucleus of the intervertebral disc to prevent its further displacement [61]. It is not a spinal cord decompression technique. It can be combined with hemilaminectomy, pediculectomy, corpectomy, and the Ventral Slot technique. The manipulation is performed for diseases of intervertebral discs of type I and II using the Ventral Slot method in the cervical spine. In case of disc protrusion, the intervertebral spaces are fused at the level of the second cervical to the sixth cervical vertebrae (C2 - C6), the eleventh thoracic to the twelfth thoracic vertebrae (T11 - T12) to the third lumbar to the fourth lumbar vertebrae (L3 - L4). It is important that access to the C6 - C7 and T10 - T11 vertebrae is too difficult [72].

The provision of care is based on the RAT principle: R for recognise, A for assessment, T for treatment.

A foramenectomy is a surgical procedure that allows access to the articular surfaces of the vertebrae and intervention at the level of the intervertebral foramen in case of spinal cord compression with possible resection of osteophytes that cause nerve compression.

Corpectomy is an operation in which the vertebral body and part of the intervertebral disc are removed.

Facetectomy is the removal of articular surfaces [129]. The manipulation is performed by compressing the nerve root to identify the appropriate intervertebral foramen with a drill or Luer forceps to remove the articular processes.

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1.6. Conclusion to Section 1

Taking into account the above data, it can be concluded that veterinary vertebrology and veterinary neurology are developing somewhat slower than other areas of practical surgery. Significant developments and accumulation of new schemes for diagnosing spinal cord and spinal cord injuries, algorithms for providing emergency care, belong to well-known scientists and practitioners in the workplace who are constantly engaged in clinical reception and management of patients in the neurosurgical area of veterinary medicine. Undoubtedly, the variety of existing diagnostic and treatment methods is not widespread enough in the practice of veterinary medicine due to the difficulty in conducting high-quality diagnostics, providing qualified care to patients with spinal injuries, and the still low availability of tomographic diagnostic methods in everyday practice. Surgical methods of treatment already include at least 15 to 20 different variations with the necessary neurosurgical equipment to perform early decompression of the spinal cord from spinal nerve compression, compression of the spinal cord itself by parts of the nucleus pulposus and remnants of the torn fibrous ring, as well as elimination of compression caused by explosive fractures of vertebral bodies and various multiethiological factors that have caused damage to the animal's axial skeleton.

Since the nervous tissue has little ability to restore damaged spinal nerves, damage to their roots, spinal cord compression with its damage, the aim is to set goals to study the main pathophysiological processes that occur after a spinal cord injury and to perform decompression and stabilisation surgery in dogs to restore the function of the motor apparatus while maintaining activity.

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SECTION 2
RESULTS OF OUR OWN RESEARCH

2.1. Testing methods for diagnosing spinal injuries in dogs

The methods we used to diagnose spinal cord and spinal cord injuries in dogs included a general clinical examination, as well as special research methods, such as orthopaedic, neurological, myelographic, radiological, cytological and biochemical examination of cerebrospinal fluid, haematological and tomographic.

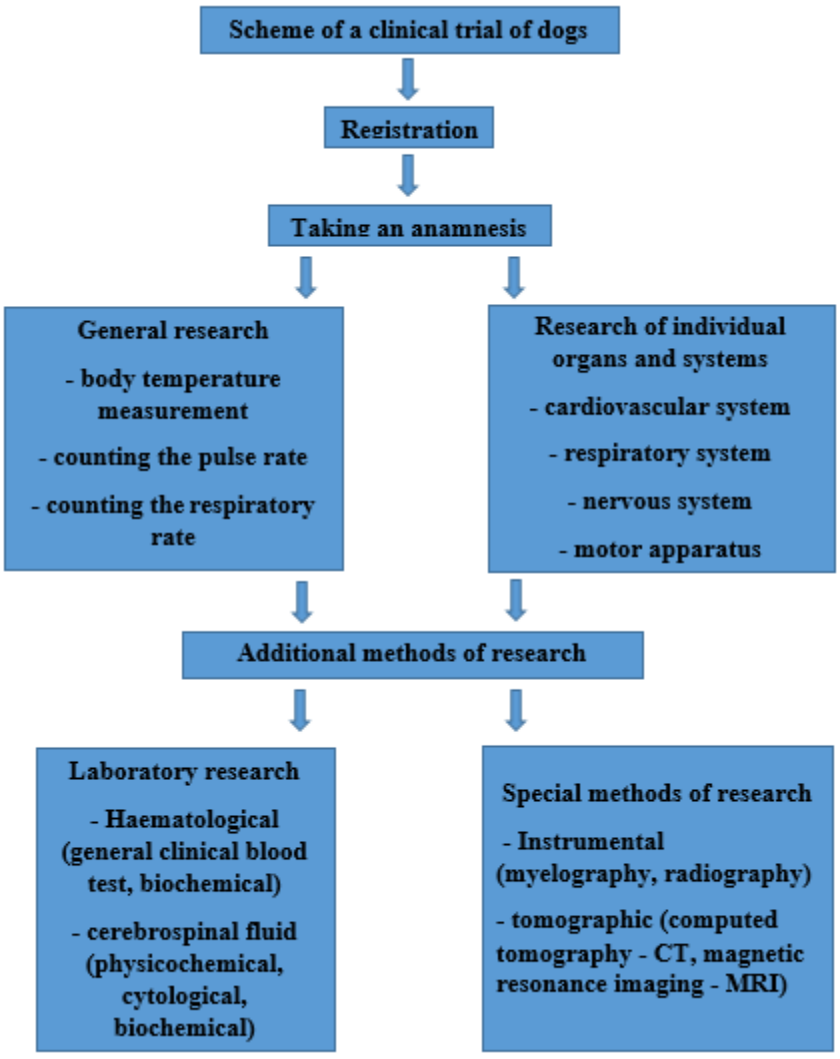

Fig. 3.1 - Scheme of a clinical trial of dogs

The vast majority of diagnostic methods are available in veterinary clinics, where laboratories are adequately equipped with the appropriate material and technical facilities, including X-ray rooms. The use of modern diagnostic techniques, neurological scales and protocols for orthopaedic and neurological examinations

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allows us to establish the location of the spinal column injury in a timely and accurate manner and provide timely emergency care.

The diagnostic process requires a comprehensive examination of the sick animal to determine the extent of damage to the spine and spinal cord, its membranes and nerve roots. Based on the data obtained and the location of the spinal injury, it is possible to prescribe a course of conservative and surgical treatment, as well as determine the prognosis for the near future when the patient is in hospital. During the admission of the animal in the veterinary clinic, the following proposed scheme of research methods in dogs was performed, which are shown in Fig. 3.1.

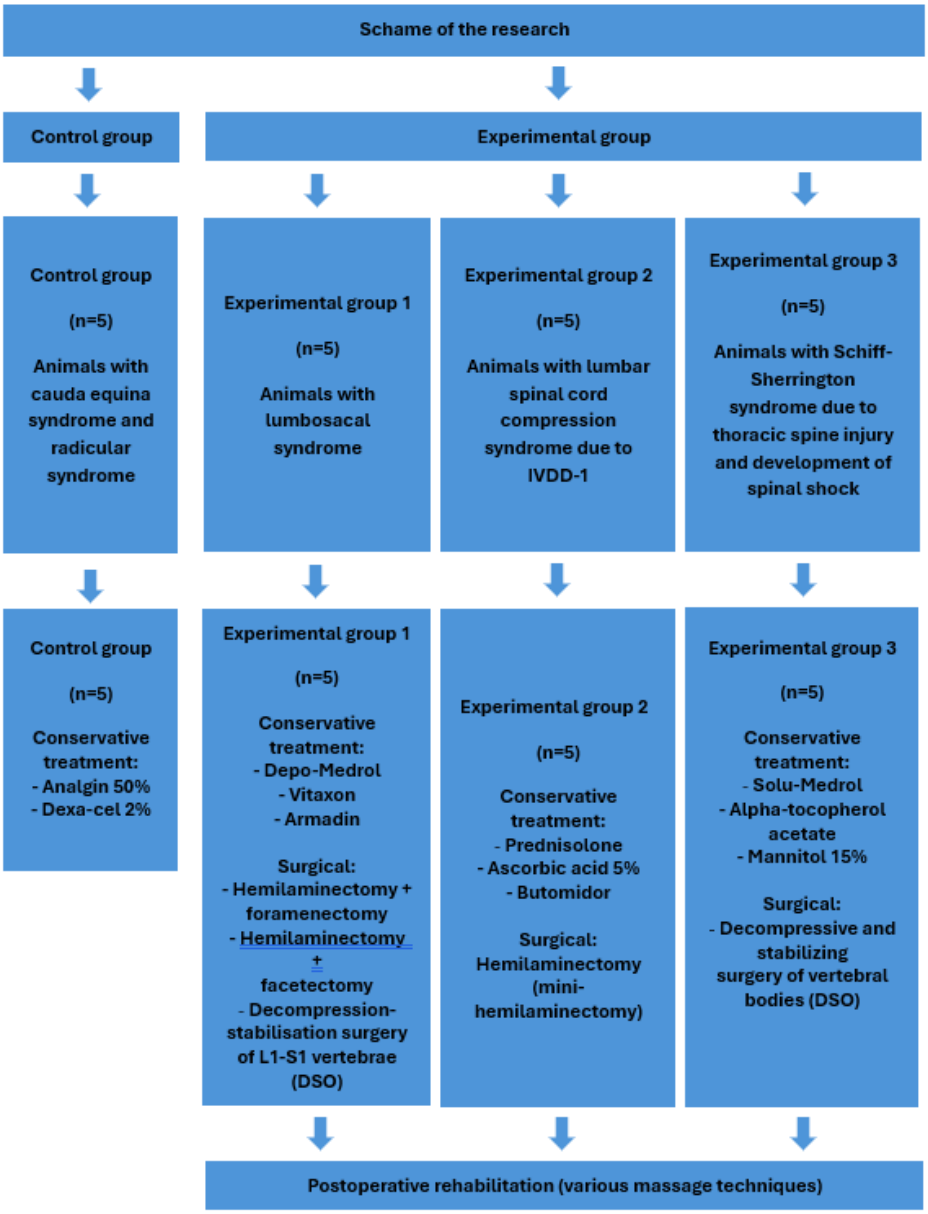

Fig. 3.2 - Scheme of the experiment in dogs

After completing a set of diagnostic measures, the next stage was the formation of control (C) and experimental groups of dogs (E1, E2, E3), n = 5 each, followed by the appointment of conservative and surgical methods of treatment (Fig. 3.2). The main condition for conducting the experiments was compliance with the proper conditions for keeping dogs in the veterinary medicine clinic, ensuring the planned studies of cerebrospinal fluid, haematological studies and timely implementation of additional diagnostic methods.

Comprehensive research was carried out in the following stages:

Stage I: general clinical trial (n = 155);

Stage II: use of instrumental and tomographic diagnostic methods;

Stage III: formation of control (C) and experimental groups (E1, E2, E3);

Stage IV: use of laboratory diagnostic methods;

Stage V: conservative and surgical treatment;

Stage VI: postoperative rehabilitation;

2.1.1. Conducting a clinical examination of dogs

Clinical examination of dogs was a prerequisite for their admission to the veterinary clinic with various symptom complexes, including orthopaedic and neurological signs. Since the injuries of the spine and spinal cord are quite numerous, and different parts of the spine can be simultaneously involved in the pathological process, the symptom complex was diverse. Recently acquired spinal cord injuries caused severe pain, spinal stiffness and stiffness of movements when moving or jumping on the floor. Comparatively advanced injuries had much less pronounced clinical signs and were manifested by moderate pain syndrome and the absence of acute neurological deficits. After obtaining physiological parameters of temperature, pulse and respiration, auscultation of the lungs and heart for the development of concomitant complications, a full orthopaedic and neurological examination of the animal was performed to determine the neurological status.

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2.1.2. Optimisation of anaesthesia in dogs during surgical treatment of animals with neurological syndromes

Before performing contrast myelography and cerebrospinal fluid cytology, dogs were anaesthetised. The animals were kept on a fasted diet for 6-8 hours and their body weight was determined by weighing. 2 to 3 hours before the planned manipulations, the preoperative profile was determined, i.e. haematological blood tests were performed to detect concomitant pathologies and inflammatory processes in a timely manner. In cases of SCI (spinal cord injury), at the early stage of clinical and neurological examination, the patient was fixed on an orthopedic board, and, if necessary, X-ray diagnostics of the corresponding spine was performed to detect complications in the form of pulmonary edema with possible development of cardiopulmonary insufficiency. To prevent additional injury during diagnostic procedures, orthopaedic pillows with fillers were used to prevent excessive pressure on the injured body part from the X-ray table.

20 to 25 minutes before the procedure, premedication with Meditin 0.1% (medetomidine hydrochloride 10 mg/mg) was performed at a dose of 0.1 to 0.2 ml per 10 kg of body weight, and as a sedative and analgesic at a dose of 0.3 to 0.8 ml/10 kg of body weight by intravenous or subcutaneous administration. In cases of severe pain syndrome, Butomidor (butorphanol in the form of hydrogen tartrate in the amount of 10 mg) was administered intramuscularly at the rate of 0.25 mg/kg body weight for analgesic effect and 0.4 mg/kg body weight for sedation in the form of intravenous administration.

Prednisolone was used subcutaneously at a dose of 1 mg/kg body weight as an anti-shock and anti-inflammatory drug. If anaesthesia was required, Zolethyl 100 (tiletamine) was administered at a dose of 7-10 mg/kg for non-invasive interventions, and 10-15 mg/kg for anaesthesia in dogs. Before the drug administration, atropine sulfate was injected at a dose of 0.1 mg/kg to reduce glandular secretion and bradycardia.

Contrast myelography was performed in 20 dogs from one control (C) and three experimental groups (E1, E2, E3). Complications in the form of an allergic reaction

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were observed in 1 dog (E2), skeletal muscle hypertonicity in 1 dog (E2), which resolved in a few minutes without the use of medications. Other patients tolerated the anaesthetic regimen described above quite well.

2.1.3. Orthopaedic examination

In the vast majority of dogs admitted to the veterinary medicine clinic for initial treatment, a rather high dependence of clinical signs on the spinal cord injury was noted. In some cases, there was a clear clinical picture indicating an injury to the musculoskeletal system, in other cases, pain syndrome in all animals without exception at the entire stage of the study indicated damage to the spine and spinal cord. Therefore, to differentiate between spinal and musculoskeletal injuries, a detailed orthopaedic examination was performed to determine the main symptom complex and its belonging to a specific traumatic group (Table 3.1).

The orthopaedic examination scheme comprised the following steps: determining the presence or absence of lameness; assessing the orthopaedic or neurological nature of the lameness; determining which limb was affected; determining the degree and nature of the lameness; determining the localisation of pain; conducting special orthopaedic tests; establishing differential diagnoses for the identified diseases of the musculoskeletal system; performing instrumental and laboratory tests to verify the diagnosis; determining further tactics of care; formulating a prognosis in the case of treatment methods (conservative or surgical).

The next step in the diagnosis of lameness was to determine its degree in points after the exercise (Table 3.2), likely as [130].

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Table 3.1 - Complex of orthopaedic examinations in the control and experimental dogs' groups (n = 5)

Animal groups		Lameness (+/-) Provisions.	Nature and extent lameness	To which. limbs He limped.	Orthopaedic test Ortholani (negative,n/p)
Control group					
1	German shepherd, 5 years old, ♂	+	intermittent	pelvic	negative
2	German Shepherd, 4 years old, ♂	+	intermittent	pelvic	negative
3	German shepherd, 9 years old, ♀	+	intermittent	pelvic	negative
4	German shepherd, 8 years old, ♂	+	intermittent	pelvic	negative
5	German shepherd, 6 years old, ♀	+	intermittent	pelvic	negative
Experimental group 1					
1	SAV, 3 p., ♀	+	shackled: 3 st.	pelvic	n/p
2	French bulldog, 7 years old, ♀	+	intermittent	pelvic	negative
3	SAV, 4 p., ♀	+	intermittent	pelvic	negative
4	German shepherd, 5 years old, ♂	+	intermittent	pelvic	negative
5	Pug, 5 years old, ♂	+	intermittent	pelvic	negative
Experimental group 2					
1	French bulldog, 4 years old, ♂	+	shackled: 2 st.	t/paresis	negative
2	Rottweiler, 7 years old, ♂	+	shackled: 2 st.	t/paresis	negative
3	German Shepherd, 7 years old, ♂	+	shackled: 3 st.	t/paresis	n/p
4	Brindle dachshund, 4 years old, ♂	+	shackled: 3 st.	t/paresis	negative
5	German shepherd, 6 years old, ♂	+	shackled: 3 st.	t/paresis	n/p
Experimental group 3					
1	Brindle dachshund, 5 p., ♂	"-" recumbent	not moving.	t/paresis	n/p
2	Brindle dachshund, 6 p., ♂	"-" recumbent	not moving.	t/paresis	n/p
3	German shepherd, 6 years old, ♂	"-" recumbent	not moving.	t/paresis	n/p
4	German shepherd, 5 years old, ♂	"-" recumbent	not moving.	t/paresis	n/p
5	French bulldog, 8 years old, ♀	"-" recumbent	not moving.	t/paresis	n/p

Note: *t/paresis - tetraparesis, n/movement - does not move; n/p - not performed.*

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Table 3.2 - Degree of lameness in dogs according to Innes

Degree of lameness	After loading	Degree of painfulness
0 - no lameness	Absence of lameness	No pain during joint manipulation
1 - mild lameness	Walking is mostly without lameness, with slight lameness after prolonged exertion. When standing, abnormal posture may not fully load the injured limb	Minor discomfort when manipulating the limb (joint)
2 - moderate lameness	Intermittent lameness that occurs after a long walk	Moderate pain during manipulation with a limb (with a joint)
3 - medium lameness	Constant limping while walking	Severe pain during manipulation
4 - severe lameness	Lack of full support, in some cases complete absence of support on the injured limb	Severe pain during manipulation

2.1.4. Neurological examination

To obtain an objective assessment of the animal's condition, a neurological protocol was used (Table 3.3), followed by determination of damage to spinal cord or relevant nerve groups. Its main indicators were examination of the head, individual cranial nerves, determination of spinal and postural reflexes, and pain sensitivity. Assessment of gait and body positioning in space were also considered valuable data. The clinical signs detected in the animal could be grouped into a neurological syndrome, which, as a rule, had a more severe course and a cautious prognosis. During the treatment period of up to 30-35 days, it was possible to draw up from 2 to 7 neurological protocols, which allowed for careful monitoring of the patient's clinical condition and timely response to changes in behaviour or detection of neurological deficits. Careful accumulation of information about the condition of dogs allows for a correct diagnosis and appropriate prescription of conservative or surgical treatment [78, 113].

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Table 3.3 - Neurological protocol

L = left, **R** = right; Y = yes, **N** = no; N/S/A/E: **N** = normal, **S** = slowed, reduced or attenuated, **A** = absent, **E** = enhanced

Examination of the head

Seizures (Y/N)	Mental status (description)
Head pressing (Y/N)	Head rotation (Y/N and direction)
Uncoordinated head movements or head tremor (Y/N)	Head lowering (Y/N and direction)

Examination of cranial nerves

	Left.	**Right**		**Left.**	**Right**
Smell (Y/N)			Threat reflex (Y/N)		
Vision (Y/N)			Average pupil size (Y/N)		
Midriaz (Y/N)			Miosis (Y/N)		
Direct pupil reflex (Y/N)			Common pupil reflex (Y/N)		
Strabismus (strabismus) and direction (Y/N)			Positional strobismus (Y/N)		
Ptosis (Y/N)			Enophthalmos (Y/N)		
Retraction of the eyeball (Y/N)			Intranasal sensitivity (Y/N)		
Atrophy of the temporalis muscle (Y/N)			Jaw tone (N/S/ A /E)		
Jaw movement range (N/S/ A /E)			Eyelid reflex, ear reflex and cheek reflex (N/S/ A /E)		
Normal nystagmus (Y/N)		(Y/N)	(Y/N)		
Position nystagmus (Y/N)			Hearing (Y/N)		
Swallowing (Y/N)			Regurgitation (Y/N)		
Change the sound (Y/N)			Stridor (Y/N)		
Atrophy of the trapezius muscle (Y/N)			Atrophy of the tongue (Y/N)		

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Gait assessment (walk, trot, canter, turns, stride, one-way walk, trolley)

Examination of the neck and thoracic extremities			**Examination of the back, pelvic limbs, anus and tail**		
	Left.	**Right**		**Left.**	**Right**
Postural reflexes					
Jumping (N/S/A)			Jumping (N/S/A)		
Conscious proprioception (N/S/A)			Conscious proprioception (N/S/A)		
Spinal reflexes					
			Knee cup (N/S/A/E)		
Biceps brachii muscle (N/S/A/E)			Calf (N/S/A/E)		
Triceps brachii muscle (N/S/A/E)			Cranial tibialis muscle (N/S/A/E)		
Radial extensor of the wrist (N/S/A/E)			Sciatic nerve (N/S/A)		
Bender (N/S/A)			Bender (N/S/A)		
Cross extension (Y/N)			Cross extension (Y/N)		
			Anal (N/S/A)		
			Tail (N/S/A)		
Miscellaneous					
	Left.	**Right**		**Left.**	**Right**
Babinski reflex (Y/N)			Babinski reflex (Y/N)		
Muscle atrophy (Y/N, localisation)			Muscle atrophy (Y/N, localisation)		
			Uncontrolled urination (Y/N)		
			Random tail movements (Y/N)		
Pain sensitivity					
Pain in the neck area (Y/N)			Pain in the neck (Y/N)		

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Surface sensitivity (Y/N)			Surface sensitivity (Y/N, localisation)		
Deep pain sensitivity (Y/N)			Trunk skin reflex (panniculitis reflex) (Y/N, localisation)		
			Deep pain sensitivity (Y/N)		

Damage location(s): _____
Severity of injury(s): mild, moderate, severe.

To determine the clinical signs of spinal cord injury, the scale of neurological disorders according to Griffiths, 1982 (classification scheme adapted from Griffiths) was used (Table 3.4):

Table 3.4

0 - normal, no pain syndrome.
1 - only pain syndrome (hyperesthesia).
2 - impairment of proprioception only (proprioceptive ataxia) or ambulatory paraparesis, the resistance function of the limb is preserved.
3 - non-ambulatory paraparesis, limb resistance function is impaired.
4 - non-ambulatory paraparesis, limb resistance function is impaired, urinary disorders (deep pain sensitivity).
5 - non-ambulatory paraparesis with the absence of deep pain sensitivity.

Table 3.5 - A set of neurological examinations in the control group (n = 5) and experimental dogs (n = 5)

	Animal groups	Scale for Griffiths (0-5 points)	Scott & McKee scale (1-6 points)	Patel reflex (+/-)	Syndrome. panic ulita (+/-)	The consciousness of the pro. (+/-)	Scale TLICS (0-10 points)
Control group							
1	German shepherd, 5 years old, ♂	2-1	2-1	+	+	+	4-4
2	German Shepherd, 4 years old, ♂	2-1	2-1	+	+	+	4-4
3	German shepherd, 9 years old, ♀	2-1	2-1	+	+	+	4-4

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4	German shepherd, 8 years old, ♂	2-1	2-1	+	+	+	4-4
5	German shepherd, 6 years old, ♀	2-1	2-1	+	+	+	4-4
Experimental group 1*.							
1	SAV, 3 p., ♀	3-0	3-n/p	+	+	+	6-0
2	French bulldog, 7 years old, ♀	3-0	2-n/p	+	+	+	4-0
3	SAV, 4 p., ♀	3-0	2-n/p	+	+	+	4-0
4	German shepherd, 5 years old, ♂	3-0	2-n/p	+	+	+	4-0
5	Pug, 5 years old, ♂	3-0	2-n/p	+	+	+	4-0
Experimental group 2*.							
1	French bulldog, 4 years old, ♂	3-2	2-4	+	+	+	5-3
2	Rottweiler, 7 years old, ♂	3-2	2-4	+	+	+	5-3
3	German Shepherd, 7 years old, ♂	3-5	2-5	+	+/-	+/-	8-4
4	Brindle dachshund, 4 years old, ♂	3-5	2-5	+	+/-	+/-	6-4
5	German shepherd, 6 years old, ♂	3-5	2-5	-	+/-	+/-	6-4
Experimental group 3*							
1	Brindle dachshund, 5 p., ♂	4-5	4-6	-	+/-	+/-	7-5
2	Brindle-coated dachshund, 6 p., ♂	4-2	4-3	-	+	-	8-4
3	German shepherd, 6 years old, ♂	4-2	4-2	-	+	-	7-4
4	German shepherd, 5 years old, ♂	4-2	4-2	-	+	-	9-4
5	French bulldog, 8 years old, ♀	4-5	4-6	-	+/-	+/-	7-5

Notes: *"+" - reaction is present, "-" - no reaction; n/p - not performed; * (E1, E2, E3) - numerator (1 day of administration), denominator - (30 days of administration).*

Accordingly, taking into account the gradation of the scale and the results

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obtained, determining the presence of a complex of reflexes, such as flexion, pannicular, knee, in particular, conscious proprioception and pain sensitivity, aims to more accurately determine the location of spinal cord injury (Table 3.5).

To assess the degree of neurological disorders, the scale by Scott H. W., McKee W. M. was used (Table 3.6):

Table 3.6

1. Pain reaction (the dog does not jump), slight deficit of proprioception with preservation of the ability to walk.
2. Pain and paresis with preservation of the ability to walk.
3. Paresis with little or no ability to walk.
4. Lack of motor function (paraplegia), with preservation of deep pain sensitivity.
5. Absence of motor function (paraplegia), absence of deep pain sensitivity for less than 48 hours.
6. Absence of motor function (paraplegia), absence of deep pain sensitivity for more than 48 hours.

Taking into account the requirements for orthopaedic research, the following reflexes, symptoms, syndromes and reactions were determined by the following methods.

The cross extensor reflex was performed in a dog with fixation on its side to check the conscious sensation of pain in the pelvic limb. Interpretation of the results: when a positive extensor reflex was obtained, the stimulated limb was flexed, and the opposite limb was extended. This indicates diffuse damage to the spinal cord conduction pathways and an unfavourable prognosis.

The panniculitis syndrome was performed in dogs to establish the timely location of the spinal injury. Along the animal's spine, on both sides of the midline, starting from the sacral spine and moving cranially, skin twitching was induced by tingling. Interpretation of the results: the sensory element of the panniculitis reflex is segmental and involves the spinal region from the first thoracic (T1) to the third lumbar vertebrae (L3). Afferent nerve fibres correspond to the spinal segments, which allows to identify the site of spinal cord injury.

The patellar reflex of the pelvic limb indicates the condition of the femoral (spinal segments L4 - L6) and sciatic (spinal segments L6 - S1) nerves. The study was

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used to assess the integrity of her main nerves (cutaneous, muscular, median, ulnar, radial) and spinal segments of the first cervical (C1) - second thoracic vertebrae (T2).

Conscious proprioception allows to identify early clinical signs of spinal cord injury. The reaction was determined on all limbs. A sheet of paper was placed under one of the limbs, which was then gradually moved to the side. Interpretation of the results: if the dog could not move the limb to its original position on its own, this indicated spinal cord injury.

The approved scheme of neurological examinations allows timely detection of the location of the injury, the severity of the spinal cord injury, and the establishment of causal factors.

2.1.5. Myelographic examination

All animals examined for the presence of neurological syndromes were carefully examined with the determination of basic physiological parameters: temperature, pulse and respiration. After weighing, the animals were administered symptomatic and sedative drugs to ensure a high-quality study. Particularly difficult patients with neurological syndromes (Cauda equina syndrome, radicular syndrome, lumbosacral syndrome) were administered analgesics and glucocorticosteroids through an intravenous catheter. According to the protocol of the neurological examination, measures were taken to restrict the movements of each patient to prevent additional damage to the spine and spinal cord by keeping them in a cage to ensure a state of hypodynamia [113, 131].

It is advisable to use a separate syringe with a needle for administration of radiopaque contrast agent and not to mix it with other drugs. Prior to myelography, blood was taken to prevent errors in the results of biochemical tests to determine the concentration of plasma enzymes such as AST, ALT, amylase, GGT, alkaline phosphatase, etc.

Myelographic examination was performed to identify the location and degree of spinal cord compression. To achieve the expected result, the contrast agent was injected into the occipital-atlas and lumbar spine.

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For contrasting the subarachnoid space, an iodine-containing non-ionic water-soluble radiopaque solution of Tomohexolum with an iodine content of 240 to 300 mg/ml was used. The amount of solution injected depended on its concentration and the size of the dog. Small dogs (pug, French bulldog, short-haired dachshund) were injected with 0.8 to 3.0 ml of high-contrast Tomohexol 300, respectively, and large dogs (German shepherd, Rottweiler, Central Asian shepherd) with 4.0 to 6.0 ml of Tomohexol 240 (Fig. 3.3) [31, 32, 68].

A prerequisite for myelography was to heat the contrast agent to a temperature of 30-35 degrees to avoid unwanted side effects [72, 74].

All animals were preliminarily anaesthetised to prevent technical errors that could occur during the work (subsection 3.1.2).

It is worth noting that general anaesthesia is a contraindication for contrast myelography in the presence of clinical signs of atlanto-axial instability, infectious diseases, meningitis, intracranial hypertension, spondylitis and spondyloarthritis, and allergic reactions to iodine-containing drugs.

In the case of *occipito-atlantic injection of* contrast medium, dogs were placed in a lateral position, maintaining the distance from the back to the table at the same level by placing pillows under the body. The head was bent downwards, the ears were pulled forward to open the occipito-atlantic space, and the surgical field was prepared with antiseptic skin treatment. The anatomical landmarks for the injection site were the anterior edges of the atlas wings and the external occipital ridge. The needle insertion site is a depression between these three structures. The SpinoCan R spinal needle with a stylet was used to carefully pierce the skin, subcutaneous tissues and muscles until the membrane (yellow fascia and arachnoid membrane) was felt to be overcome. The stylet was removed from the needle to check the presence of cerebrospinal fluid and returned if the needle needed to be deepened further. If the correct needle insertion was observed, a contrast agent was injected into the CSF (cerebrospinal fluid) of the spinal cord, and a series of X-rays were taken [113].

The unevenness of the ventral column above the interdisc spaces and the bend at the level of the second cervical to third cervical vertebrae (C2 - C3) noted in the

cervical region are physiological norms.

In the case of CR insertion into the *lumbar* spine, dogs were placed in a supine or side position, with the pelvic limbs pulled forward to provide access to the lumbar spine. After preparation of the surgical field, the spinous processes were palpated at the level of the fourth to fifth lumbar vertebrae (L4 - L5) or the fifth to sixth lumbar vertebrae (L5 - L6). After that, a 20-22-gauge spinal needle with a mandrel was inserted between the vertebrae at a caudal angle of 60-90 degrees to the skin surface. When the needle enters the epidural space, a feeling of "failure" occurs.

When the needle is in the dural space, the solution is injected easily. "Tomohexol is heavier than cerebrospinal fluid, so the dog's head should be higher than the sacrum. The needle was gently advanced until it came into contact with the bone at the bottom of the spinal canal and then moved forward or backward until it entered the CSF (cerebrospinal fluid) of the spinal cord. The mandrel was removed and CSF (cerebrospinal fluid) was obtained by gentle aspiration. Contrast fluid was slowly injected. After that, X-rays of the spine were taken in the lateral and VD projections. Extradural compression was determined by the absence of a contrast column in the area of injury, with the dura sac narrowing to the LSR (lumbosacral region) and ending at the level of the sixth lumbar vertebrae - the third sacral vertebrae (L6 - S3).

In all dogs undergoing myelography, no complications were detected after the introduction of CR into the occipital-atlas and lumbar spine.

In total, myelographic examination was performed in 20 cases, including the control group (C, n = 5) and experimental groups (E1, E2, E3, n = 5 each). In 5 experimental animals (group E3) with Schiff-Sherrington syndrome, characterised by acute spinal cord injury and impaired integrity of the CSF (cerebrospinal fluid) and spinal cord, CSF (cerebrospinal fluid) leakage through damaged tissues was observed (Table 3.7).

Myelographic studies show that contrast myelography is a technically simple diagnostic method in everyday practice and allows to determine changes in spinal cord SAP in various clinical forms of spinal cord injury. A complete examination takes up to 20-30 minutes. The main disadvantage of the manipulation is the temporary

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deterioration of neurological symptoms, which does not allow for a full differential diagnosis.

In cases of mild spinal cord compression, a partial stop of contrast medium distribution in the subarachnoid space was found. This indicates that spinal cord compression is observed in the corresponding area, while this effect was not observed during myelography in other animals due to free spread of Tomohexol from CSF (cerebrospinal fluid).

Fig. 3.3 - Tomohexol-300 radiopaque contrast agent for myelographic examinations

In case of vertebral body fractures in the lumbar spine, stenosis of the subarachnoid space border was detected, which resulted in disruption of the contours of radiopaque columns running parallel to each other (dorsally and ventrally).

2.1.6. X-ray examination

X-ray examinations were performed to determine the nature of vertebral body fractures with further assessment of the degree of bone callus development using the BATEL-1 X-ray machine with Digipax software.

The X-ray examination was performed depending on the location of the spinal injury in three projections, namely: ventro-dorsal (VD), lateral and frontal (Figs. 3.4,

3.5).

The obtained radiographs were evaluated according to the following criteria:

1. Clarity of the X-ray image and compliance with the correct stacking.

2. Examination of the vertebrae for fractures, displacements, curvatures, and shortening of the bodies.

3. Determination of spinal curvature with subsequent rotation of the vertebral bodies.

4. Determination of intervertebral spaces in the cervical, thoracic and lumbar spine.

5. Evaluation of symmetry and shape of exostoses, their size, and osteophyte formation.

6. Determination of the shape, width of the spinal canal and the presence of mineralised inclusions in the epidural space.

7. Determination of pathological changes in the surrounding spinal tissues.

The results of the X-ray examination are presented in Table 3.7.

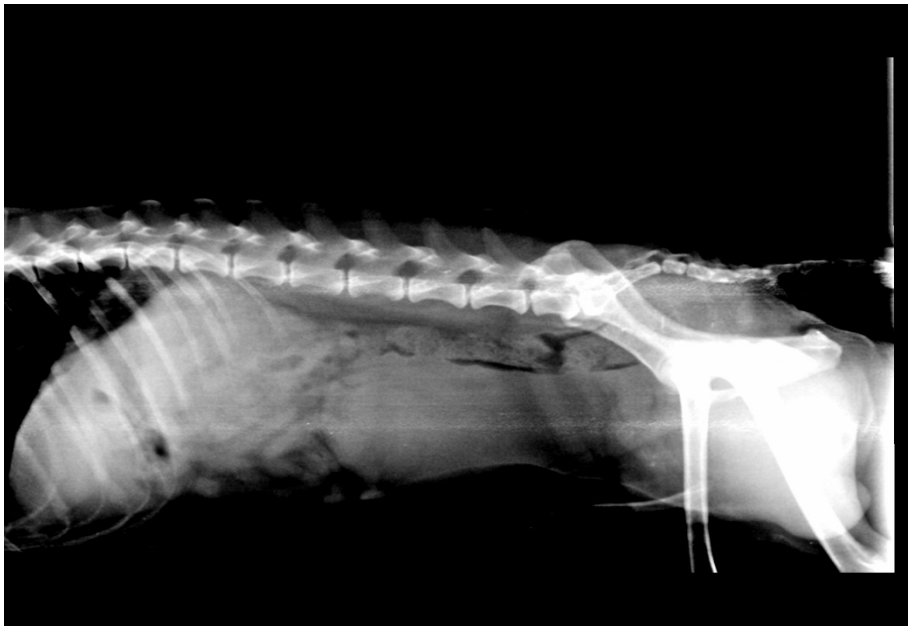

Fig. 3.4 - Radiograph of the lumbar spine in a German Shepherd dog, 5 years old, named John, ♂ (E1). Lumbosacral stenosis at the level of L7 - S1. Lateral projection (the image was taken in the negative). There is a narrowing of the distance between the L7 vertebrae in relation to the S1 vertebrae

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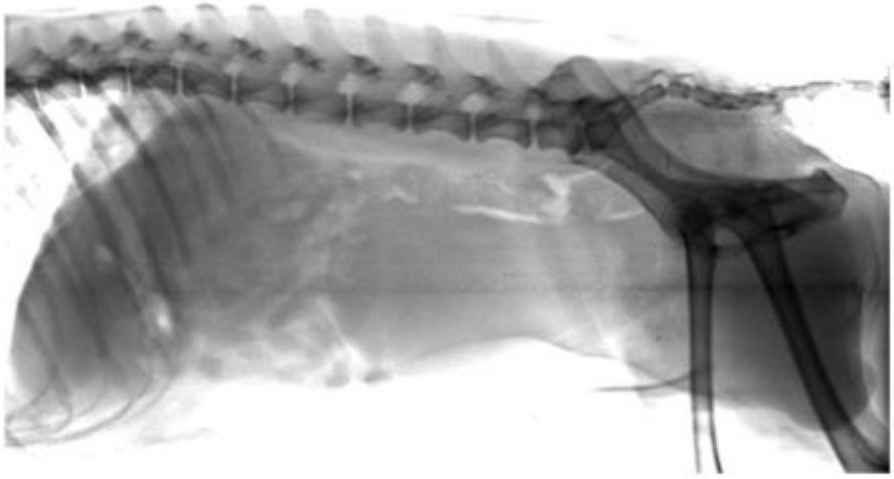

Fig. 3.5 - The animal from Fig. 3.4 (E1). Lateral projection (positive view)

Table 3.7 - A set of radiological and myelographic examinations in the control group (n = 5) and experimental dogs (n = 5)

Animal groups		Myelography (+)	Stenosis IVS (+/-)	Violation of the CA due to SCC	Diagnosis of concomitant pathologies
Control group					
1	German shepherd, 5 years old, ♂	+	+	+	LSS stenosis
2	German Shepherd, 4 years old, ♂	+	+	+	LSR stenosis
3	German shepherd, 9 years old, ♀	+	+	+	LSR stenosis
4	German shepherd, 8 years old, ♂	+	+	+	LSR stenosi
5	German shepherd, 6 years old, ♀	+	+	+	LSR stenosis
Experimental group 1					
1	SAV, 3 p., ♀	+	+ (L7-S1)	+ (L2)	Bone fragments LSR stenosis
2	French bulldog, 7 years old, ♀	+	+ (L7-S1)	+	Cyst (L7)

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Animal groups		Myelography (+)	Stenosis IVS (+/-)	Violation of the CA due to SCC	Diagnosis of concomitant pathologies
3	SAV, 4 p., ♀	+	+ (L7-S1)	+	LSR stenosis
4	German shepherd, 5 years old, ♂	+	+ (L7-S1)	+	LSR stenosis
5	Pug, 5 years old, ♂	+	+ (L7-S1)	+	LSR stenosis
Experimental group 2					
1	French bulldog, 4 years old, ♂	+	-	+ (L5)	-
2	Rottweiler, 7 years old, ♂	+	-	+ (L2-L3)	-
3	German Shepherd, 7 years old, ♂	+	-	+ (L1-L2)	Exostosis
4	Brindle dachshund, 4 years old, ♂	+	-	+ (T7-T8)	Compression of the SC
5	German shepherd, 6 years old, ♂	+	-	+	Compression of the SC
Experimental group 3					
1	Brindle dachshund, 5 years old, ♂	+	-	Leakage of the CA	Bone fragments
2	Brindle-coated dachshund, 6 p., ♂	+	-	Leakage of the CA	-
3	German shepherd, 6 years old, ♂	+	-	Leakage of the CA	Bone fragments
4	German shepherd, 5 years old, ♂	+	-	Leakage of the CA	-
5	French bulldog, 8 years old, ♀	+	-	Leakage of the CA	Bone fragments

Notes: CA - contrast agent, IVS - intervertebral space,
LSR - lumbosacral region; SCC - spinal cord compression

To prevent technical errors that could occur during the diagnostic process, the dogs were administered with drugs to ensure a state of light or moderate sedation (subsection 3.1.2).

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2.1.7. Cytological and biochemical examination of cerebrospinal fluid

During the treatment of dogs with SCI (spinal cord injury), complications often occur in the form of hyperthermia, intoxication, severe dehydration and the appearance of a neurological status that was not characteristic of the initial treatment period. In this case, there may be a suspicion of myelitis, encephalitis, spinal shock, sepsis or viral infection. For this purpose, a cytological examination of the cerebrospinal fluid is performed to count the total number of cells and their differentiation. The examination was performed within 30 to 60 minutes after the puncture. A Fuchs-Rosenthal chamber was used to count the number of leukocytes.

During macroscopic examination of the cerebrospinal fluid, its colour, transparency and relative viscosity were determined. The presence of protein and glucose was determined by biochemical methods (Tables 3.8-3.11). In doubtful cases, when the data obtained did not provide an indication of the presence of signs of a particular disease, the cerebrospinal fluid was examined in the laboratory by enzyme-linked immunosorbent assay (ELISA).

The indication for the puncture was suspected CNS disease, even if the results of haematological blood tests were within the reference range. It is best to perform CSF sampling when the animal is anaesthetised before contrast myelography [63, 64, 91].

Contraindications for puncture: neurological disease caused by acute trauma, metabolic disorders, intervertebral disc diseases accompanied by increased pressure in the subarachnoid space. Elevated intracranial pressure (intracranial hypertension), tentorial hernia, thrombocytopenia (PLT < 50 x 109/L).

In life-threatening conditions, it was necessary to perform tracheal intubation with subsequent oxygen supply through an oxygen concentrator.

A rather significant diagnostic measure in dogs of the control (C) and experimental groups (E1, E2, E3) was the examination of cerebrospinal fluid on the first and thirtieth day of observation. The fluid was sampled in the occipito-atlantic space in the amount of 1.5 to 3 ml, depending on the body weight of the animal, in compliance with the proper rules of asepsis and antisepsis. The main parameters in the diagnosis were the determination of the physicochemical properties of the

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cerebrospinal fluid, cytological and biochemical examination (Tables 3.8-3.11).

In dogs of the control group, the colour of the cerebrospinal fluid was clear to slightly opalescent, indicating the presence of minor blood impurities, namely erythrocytes. Slight turbidity is also due to the presence of erythrocytes, but is more permanent after centrifugation. During the study period, no pathological changes in the physicochemical properties of the fluid, including relative density, were detected.

Microscopic examination revealed a complete absence of eosinophils in the CSF (cerebrospinal fluid) of all dogs during the two-time sampling. The leukogram showed the presence of the permissible number of elements within the established norm and no excess of 5 cells/mL was noted, indicating the absence of a viral infection in the body. In particular, the number of neutrophils also did not exceed the established reference standard, indicating the absence of neutrophilic pleocytosis and the development of meningitis.

During the study period, no hyperproteinuria was detected in dogs, which is explained by the absence of an exacerbation phase of the inflammatory process in the form of arachnoiditis and subarachnoid haemorrhages with degenerative changes in older dogs. In two cases, moderate hypoproteinarchy was noted, which is probably an indication of intracranial hypertension. The biochemical parameters of glucose and urea levels remained within the physiological norm and the absence of azotemic meningoencephalitis, subarachnoid haemorrhage, and uremic syndrome was noted throughout the observation period (Table 3.8).

In the animals of **the first** experimental group, the transparency of the cerebrospinal fluid was mostly slightly cloudy to opalescent, which may indicate close passage of the needle through small blood vessels and the ingress of red blood cells into the samples. If the blood impurities are accidental and obtained during centesis, the liquid column above the sediment is completely colourless and the red blood cells are fresh, unchanged. A grey and slightly cloudy colour is caused by the presence of cellular elements in the samples and their number should be carefully controlled by repeated centrifuges. The relative density of the fluid did not change during the study period and remained within the reference range.

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Table 3.8 - Physicochemical, cytological and biochemical tests cerebrospinal fluid in dogs of the control group (n = 5)

Indicators/ dog breeds	German shepherd, male, 5 p., ♂	German shepherd, male, 4 p., ♂	German shepherd, female, 9 p., ♀	German shepherd, male, 8 p., ♂	German shepherd, female, 6 p., ♀	Reference norms [63, 134]
Day 1 (numerator) and day 30 (denominator) (control group)						
Physicochemical properties of CSF (macroscopic examination)						
Colour/. transparency	transparent	grey	slightly opalescent	slightly opalescent	transpare-nt	Colourless/ transparent
	slightly opalescent	transparent	slightly cloudy	slightly cloudy	slightly sediment	
Relative density	1.006	1.007	1.006	1.006	1.006	1.006-1.008 g/ml
	1.006	1.006	1.008	1.008	1.007	
Cytological examination (leukogram) of CSF (microscopic examination)						
Eosinophils	missing	missing	missing	Missing	missing	Absent
	missing	missing	missing	Missing	missing	
Lymphocytes	1-2	2-3	3-4	2-3	4-5	2-4 cells per 1 µl.
	2-3	2-3	1-2	3-4	2-3	
Monocytes	1-2	1-2	2-3	2-3	2-3	1-3 cells per 1 µl.
	2-3	2-3	2-3	2-3	3-4	
White blood cells	missing	missing	1-3	2-4	1-3	0 to 3-8
	1-3	missing	2-4	2-4	3-5	
Biochemical examination of CSF						
Protein (g/l)	0.23	0.21	0.21	0.18	0.26	0.22-0.33
	0.25	0.17	0.24	0.20	0.25	
Glucose (mmol/l)	2.77	3.14	4.17	4.02	3.82	2.5-4.44
	2.82	3.38	4.24	4.42	3.64	
Urea (mmol/l)	1.16	1.12	2.04	1.74	2.04	1.0-3.3
	1.34	1.24	2.23	1.99	2.37	

Cytological examination of the cerebrospinal fluid revealed the absence of eosinophils in all dogs during the observation period. The number of lymphocytes on day 1 of the study in dogs was within normal limits, and on day 30 their number

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increased by 50-75% compared to the upper limit of normal, indicating progression of changes in bone tissue, including the development of polyneuritis. On the contrary, the number of monocytes compared to lymphocytes decreased by up to 25% compared to the upper limit of the norm, which indicates the presence of a temporary inflammatory process. As a result, in three dogs of the first experimental group, an increase in the number of leukocytes by 15% relative to the established limit of the norm was noted, and the presence of an inflammatory process was confirmed.

According to the biochemical study, the amount of protein was within the lower to the average statistical limit of normal. No hypoproteinarchy or hyperproteinarchy was observed during the entire observation period. Glycoarchy in experimental dogs was within the reference limits and is an indicator of physiological normality. It is worth noting that the urea concentration also fluctuated in a small amount and did not exceed the established values. Taking into account the fact that the animals of the first experimental group had lumbosacral syndrome with combined SCI, no disorders of the urinary system or development of uremic syndrome were detected (Table 3.9).

On day 1 of the study, macroscopic examination of animals in **the second** experimental group revealed changes in the fluid from slightly cloudy to cloudy, which causes signs of inflammation at the beginning of the diagnostic process. As a result, on the 30th day of the study, 80% of dogs showed xanthochromia, which has significant changes in the colour of the fluid from slightly cloudy to sharply cloudy and pronounced yellowish, pinkish and brownish colour. This condition is most often caused by cerebrospinal fluid dysregulation, significant intervertebral disc disease in the form of protrusion with subsequent development of the ischaemic process. As a result, in the same dogs, an increase in the relative density of CSF by 25-75% relative to the upper limit of the norm was noted, indicating the accumulation of a large number of cellular elements.

Microscopic examination revealed the absence of eosinophils in 40 % of dogs in the samples, and in 60 % of experimental animals single shaped blood elements were observed, which, obviously, may indicate the development of myelitis over time. The number of lymphocytes on day 1 in all dogs was 25-75 % higher than the upper limit

Table 3.9 - Physicochemical, cytological and biochemical studies cerebrospinal fluid of the first experimental group in dogs (n = 5)

Indicators/ dog breeds	SAV, Bitch, 3 yrs, ♀	French. Bulldog, female, 7 years old, ♀	SAV, Bitch, 4 yrs, ♀	German shepherd, male, 5 p., ♂	Pug, male, 5 p., ♂	Reference norms [63, 134]
Day 1 (numerator) and day 30 (denominator) (study group 1)						
Physicochemical properties of CSF (macroscopic examination)						
Colour/. transparency	grey	slightly opalescent	slightly cloudy	opalescent	grey	Colourless/ transparent
	opalescent	slightly cloudy	opales-ent	slightly opalescent	slightly cloudy	
Relative density	1.008	1.006	1.007	1.006	1.006	1.006-1.008 g/ml
	1.006	1.007	1.007	1.008	1.007	
Cytological examination (leukogram) of CSF (microscopic examination)						
Eosinophils	missing	missing	missing	Missing	missing	Absent
	missing	missing	missing	Missing	missing	
Lymphocytes	2-3	3-4	2-3	3-4	3-4	2-4 cells per 1 µl.
	5-6	6-7	5-6	5-6	4-5	
Monocytes	2-3	1-2	1-2	1-2	2-3	1-3 cells per 1 µl.
	3-4	3-4	3-4	2-3	1-2	
White blood cells	4-6	5-7	4-6	5-7	5-7	0 to 3-8
	3-5	7-9	6-8	7-9	8-10	
Biochemical examination of CSF						
Protein (g/l)	0.26	0.24	0.30	0.25	0.26	0.22-0.33
	0.27	0.31	0.25	0.27	0.29	
Glucose (mmol/l)	2.77	3.04	2.81	3.24	3.96	2.5-4.44
	2.98	3.36	3.07	3.67	4.24	
Urea (mmol/l)	2.94	2.65	3.24	2.21	1.94	1.0-3.3
	3.22	2.91	3.01	2.87	2.27	

of the norm, and on day 30 by 200-300 %, respectively, which is explained by the development of lymphocytic pleocytosis. For comparison, the number of monocytes on day 1 was significantly increased by 25% compared to the upper limit of normal in 60% of dogs, and on day 30 these figures increased to 250%, respectively, which, in turn, also does not exclude the development of myelitis. On day 1, an increase in the

number of leukocytes by 100 - 350 % was recorded in the samples, and on day 30, these values ranged from 275 % to 575 %, respectively, which causes the development of subarachnoid haemorrhages.

The protein concentration in fluid samples on day 1 did not differ significantly in all experimental dogs, and on day 30, it increased by 1.5 times. Moderate hyperproteinuria is characteristic of subarachnoid haemorrhage and arachnoiditis. At the same time, the amount of glucose and urea is within the average physiological norm (Table 3.10).

All dogs **in the third** experimental group showed xanthochromia, where the colour of the cerebrospinal fluid ranged from pink to orange, indicating a large number of cellular elements and blood impurities. On the 30th day of the study, there were no significant changes for the better in the macroscopic assessment. As a result, the relative density was also increased by 50-75% on days 5 and 30 compared to the upper limit of normal, which may eventually lead to the development of meningitis/myelitis.

Microscopic examination on day 1 of the study revealed a complete absence of eosinophils in the CSF, while on day 30, single eosinophils were noted in 40 % of dogs, which to some extent confirms the results obtained regarding transparency and development of meningitis. The number of detected lymphocytes and monocytes exceeded the reference limits of the upper normal range by 50-100%, and characterise relative stability in the direction of increase or decrease. In this case, the pathological process is manifested by the development of polyneuritis due to SCI with the development of spinal shock. On the 1st day of diagnostics, the number of neutrophils was recorded at the level of 200 - 300% upward relative to the upper limit of the norm in 60% of dogs. On the 30th day of CSF (cerebrospinal fluid) examination, these figures were 250 - 400 %, respectively, and characterised the development of neutrophilic pleocytosis in the form of acute inflammation with subarachnoid haemorrhage.

The biochemical study of the amount of protein on days 1 and 30 revealed a slight increase in its amount in 40 % of dogs in the form of hyperproteinuria, which is associated with the development of subarachnoid haemorrhage and arachnoiditis. No

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Table 3.10 - Physicochemical, cytological and biochemical tests cerebrospinal fluid of the second experimental group in dogs (n = 5)

Indicators/ dog breeds	French. Bulldog, male, 4 p., ♂	Rottweiler, male, 7 years old, ♂	German shepherd, male, 7 p., ♂	Wool. dachshund, cob, 4 p., ♂	German Shepherd, male, 6 p., ♂	Reference norms [63, 134]
Day 1 (numerator) and day 30 (denominator) (study group 2)						
Physicochemical properties of CSF (macroscopic examination)						
Colour/. transparency	slightly cloudy	opalescent	cloudy	opalescent	cloudy	Colourless transparent
	muddy	evil/muddy	p/muddy	Muddy	p/muddy	
		Xanthochromia				
		pink	yellow.	Brown	brown	
Relative density	1.009	1.008	1.010	1.010	1.011	1.006-1.008 g/ml
	1.008	1.009	1.011	1.012	1.012	
Cytological examination (leukogram) of CSF (microscopic examination)						
Eosinophils	missing	missing	1-2	1-2	0-1	Absent
	missing	missing	2-3	2-3	2-3	
Lymphocytes	4-5	5-6	6-7	6-7	5-6	2-4 cells per 1 µl.
	7-8	7-8	9-10	11-12	12-13	
Monocytes	1-2	2-3	3-4	3-4	3-4	1-3 cells per 1 µl.
	3-4	3-4	7-8	6-7	6-7	
White blood cells	15-17	14-16	28-30	27-29	24-26	0 to 3-8
	20-22	22-24	38-40	43-45	33-35	
Biochemical examination of CSF						
Protein (g/l)	0.31	0.26	0.34	0.30	0.32	0.22-0.33
	0.37	0.32	0.41	0.46	0.47	
Glucose (mmol/l)	2.71	3.24	3.17	2.46	3.12	2.5-4.44
	2.96	3.68	3.52	2.87	3.74	
Urea (mmol/l)	1.76	1.86	1.96	2.45	2.80	1.0-3.3
	2.11	1.98	2.37	2.84	3.16	

changes in the amount of glucose were detected during the observation period, and the urea concentration had a slight increase in 40 % of dogs on day 30 with damage to the thoracolumbar spine with complications of the symptom complex of neurological syndromes (Table 3.11).

Table 3.11 - Physicochemical, cytological and biochemical studies cerebrospinal fluid of the third experimental group in dogs (n = 5)

Indicators/ dog breeds	Wool. Dachshund, male, 5 p., ♂	Wool. Dachshund, male, 6 p., ♂	German Shepherd, male, 6 p., ♂	German Shepherd, male, 5 p., ♂	French. Bulldog, bitch, 8 p., ♀	Reference norms [63, 134]
Day 1 (numerator) and day 30 (denominator) (study group 3)						
Physicochemical properties of CSF (macroscopic examination)						
Colour/. transparency	Xanthochromia					Colourless transparent
	orange.	pink	pink	pink	orange.	
	Xanthochromia					
	brown	pink	pink	pink	brown	
Relative density	1.012	1.011	1.010	1.011	1.013	1.006-1.008 g/ml
	1.013	1.011	1.011	1.012	1.013	
Cytological examination (leukogram) of CSF (microscopic examination)						
Eosinophils	missing	missing	missing	missing	missing	Absent
	2-3	missing	missing	missing	2-3	
Lymphocytes	6-7	4-5	3-4	4-5	7-8	2-4 cells per 1 µl.
	8-9	5-6	5-6	4-5	9-10	
Monocytes	4-5	1-2	2-3	3-4	5-6	1-3 cells per 1 µl.
	6-7	2-3	1-2	2-3	6-7	
White blood cells	17-19	6-8	8-10	10-12	24-26	0 to 3-8
	31-33	12-14	16-18	17-19	38-40	
Biochemical examination of CSF						
Protein (g/l)	0.35	0.28	0.27	0.29	0.37	0.22-0.33
	0.49	0.31	0.29	0.33	0.47	
Glucose (mmol/l)	2.84	2.57	2.79	3.11	4.24	2.5-4.44
	3.16	3.18	3.45	4.14	4.43	
Urea (mmol/l)	2.67	2.96	2.29	3.26	3.45	1.0-3.3
	3.02	3.36	2.84	3.07	3.64	

Abbreviations: s/muddy - slightly muddy; s/opal - slightly opalescent; r/muddy - sharply muddy; opalescent; orange. - orange.

Summarising the data obtained, it can be concluded that laboratory examination of cerebrospinal fluid is of great diagnostic value. Since macroscopic assessment provides an opportunity to evaluate physicochemical properties, including

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transparency and colour parameters, microscopic and biochemical analysis aims to establish the presence of neutrophils, which indicate a moderate or severe inflammatory process, and a leukogram that characterises the nature of the disease and the development of concomitant complications in the form of neuritis, meningitis or myelitis.

The ratio of biochemical profile indicators is a rather important element of diagnostics, as early diagnosis of uremic syndrome, azotemic meningoencephalitis, polyneuritis is aimed at timely application of the necessary complex of medicines to stop the development of complications of the urinary and nervous system. A particularly dangerous indicator is the presence of xanthochromia, which indicates a large number of cellular elements, has significant changes in the colour of the fluid from slightly cloudy to sharply cloudy and pronounced yellowish, pinkish and brownish. This condition is most often caused by cerebrospinal fluid dysregulation, significant herniated discs in the form of protrusion with subsequent development of the ischaemic process. The phenomenon of hyperproteinuria is quite dangerous, which is associated with the development of massive subarachnoid haemorrhages and arachnoiditis. A significant increase in urea levels can contribute to the development of azotemic meningoencephalitis in dogs.

2.1.8. Haematological examination

Blood morphological parameters were determined using the Mindray BC-2800 Vet and scil Vet abc haematological analysers from Scil. For 1 test, 12 µl of whole blood is required. The analyser automatically measures 16 blood parameters within 90 seconds. Thus, a blood test was performed with the subsequent determination of the number of red blood cells, white blood cells, haemoglobin content, and leukogram output (Tables 3.22-3.25).

Blood biochemical parameters were determined using a STAT FAX 1904 PLUS biochemical analyser. For the study, whole blood was centrifuged to obtain serum and added to the reagent tubes in an amount of 30 to 100 µl by micropipetting. The test of one blood sample for 10 indicators takes 17 minutes.

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2.1.9. Tomographic examinations

Tomographic examinations were performed to determine the nature of vertebral body fractures with an assessment of the degree of displacement of fragments, dislocations and spinal cord injury using a computed tomography (CT) scanner and a 0.3 Tesla magnetic resonance imaging (MRI) scanner.

Computed tomography involves a slice thickness of 1 to 3 mm, depending on the pathological process and spinal segment, which on average during the examination allows to obtain about 100 to 250 CT scans that can be interpreted for the presence of a neurological syndrome. The diagnostic time ranged from 60 to 180 seconds.

Nonionic monomeric triiodinated water-soluble radiopaque contrast agent Tomohexol (Tomovist) was used for contrasting, which contains 240 to 350 mg of iohexol in 1 ml. The elimination period is approximately 100% in 24 hours with normally functioning kidneys after intravenous administration, and the half-life is 2 hours. The drug is intended only for diagnostic studies for contrast enhancement in computed tomography.

The following parameters are recommended for CT of the spinal column in dogs: for dogs weighing up to 5 kg - 65-100 mA, 120 kV; for dogs weighing 5-15 kg - 100 mA, 120 kV; for dogs weighing 15-25 kg - 150 mA, 120 kV; for dogs weighing 25-50 kg - 150 mA, 130 kV; for dogs weighing more than 50 kg - 150-200 mA, 130 kV.

A magnetic resonance imager with a power of 0.3 Tesla was used for the work, which makes it possible to obtain high-quality MR images. The time for the examination is 25-30 minutes, in other cases it can reach 40 minutes, depending on the location of the injury. Correct animal positioning ensures high-quality results on tomograms in the segmental and sagittal planes.

When using a T1-weighted proton MRI scanning sequence, gadolinium ion-induced shortening of the spin-lattice relaxation time (T1) of atomic nuclei excitation results in increased signal intensity and contrast in certain tissues.

MRI diagnostics was used to examine the spinal cord, determine the presence of intervertebral disc herniation, detect scar tissue and a possible area of stenosis (Figs. 3.6, 3.7; Table 3.12).

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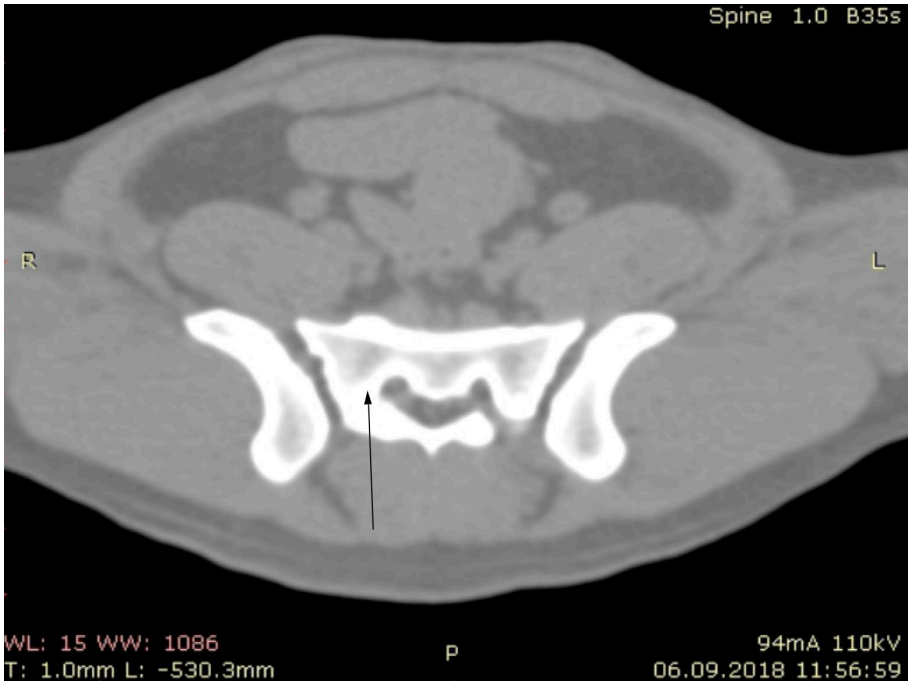

Fig. 3.6 - MR imaging of the lumbosacral spine at the level of L7 - S1 in the segmental plane in a 7-year-old French bulldog, nicknamed Teddy (E1). The arrow indicates the area of stenosis

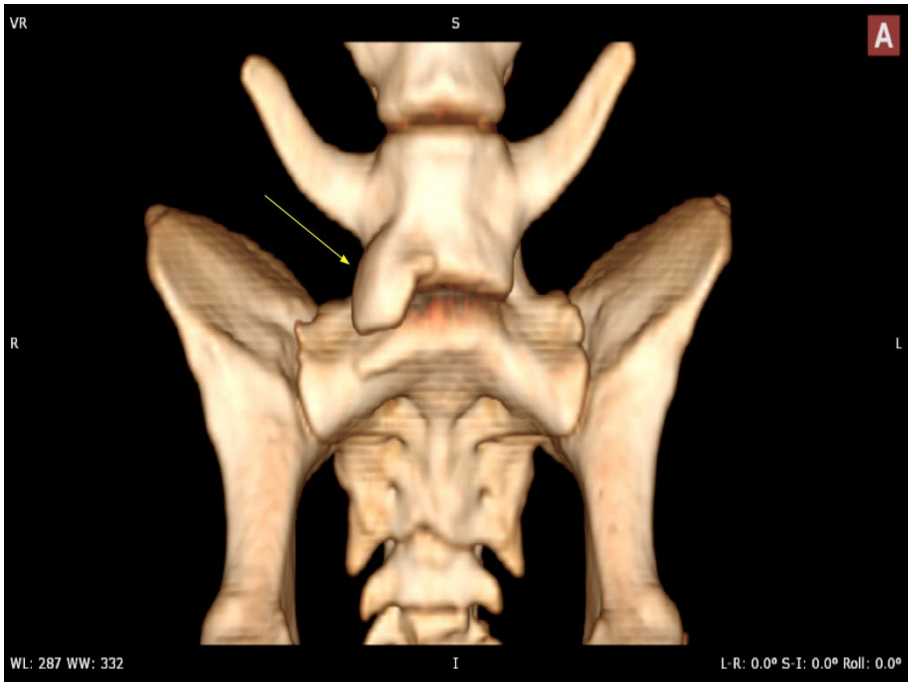

Fig. 3.7 - MR imaging of the lumbosacral spine at the level of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1) with 3D visualisation in a 7-year-old French bulldog, Teddy. Arrow indicates stenosis

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Table 3.12 - Complex of tomographic examinations in the control group (n = 5) and experimental dogs (n = 5)

Animal groups		Diseases SC and SN (MRI)	Compression fractures (CT scan)	IVD disease according to Hansen (MRI)
Control group				
1	German shepherd, 5 years old, ♂	compression of the SN	-	-
2	German Shepherd, 4 years old, ♂	compression of the SN	-	-
3	German shepherd, 9 years old, ♀	compression of the SN	-	-
4	German shepherd, 8 years old, ♂	compression of the SN	-	-
5	German shepherd, 6 years old, ♀	compression of the SN	-	-
Experimental group 1				
1	SAV, 3 p., ♀	compression of the SN	at the (L2) level	-
2	French bulldog, 7 years old, ♀	compression of the SN	-	-
3	SAV, 4 p., ♀	compression of the SN	-	-
4	German shepherd, 5 years old, ♂	compression of the SN	-	-
5	Pug, 5 years old, ♂	compression of the SN	-	-
Experimental group 2				
1	French bulldog, 4 years old, ♂	(L1-L2) Contusion	-	+ (type I) (L5)
2	Rottweiler, 7 years old, ♂	(L1-L3)	-	+ (type I) (L2-L3)
3	German Shepherd, 7 years old, ♂	(L1-L3)	(L2)	+ (type I) (L1-L2)
4	Brindle dachshund, 4 years old, ♂	(L1-L3) Myelitis	-	+ (type I) (T7-T8)
5	German shepherd, 6 years old, ♂	partial break	-	+ (type I)
Experimental group 3				
1	Brindle dachshund, 5 p., ♂	partial break	-	+ (type II)
2	Brindle dachshund, 6 p., ♂	compression of the SC	-	+ (type II)
3	German shepherd, 6 years old, ♂	compression of the SC	(T4-T5)	+ (type II)
4	German shepherd, 5 years old, ♂	partial break at the (L1-L2) level	-	+ (type II)
5	French bulldog, 8 years old, ♀	compression of the SC	-	+ (type II) (L2)

Notes: IVD - intervertebral disc, SC - spinal cord, SN - spinal nerves

Tomovist, a paramagnetic contrast agent containing 469 mg of dimeglumine gadopentetate per 1 ml, was used to contrast the spinal cord tissue. The half-life is on average 83% within 6 hours after intravenous administration.

In the range from 0.14 to 1.5 T, the recommended doses do not depend on the

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magnetic field strength.

MRI can diagnose diseases of the central nervous system, compression injuries of the spinal cord, and brain damage. The method allows for very clear visualisation of the anatomical structure of intervertebral discs, the degree of their protrusion and extrusion, partial or complete spinal cord tear, and spinal cord swelling. For a more accurate diagnosis, a multi-plane reconstruction can be performed based on the interpretation of an MRI scan. Intervertebral discs are well visualised on MRI and have a "glowing" effect. To prevent technical errors that could arise during the diagnostic process, the dogs were administered drugs to ensure a state of severe sedation and anaesthesia (subsection 3.1.2).

2.2. Clinical characteristics of neurological syndromes in acute spinal cord and spinal cord injuries

The total number of examined dogs with spinal cord and spinal cord injuries (n = 155) is shown in Tables 3.13-3.14 and Figs. 3.8-3.11.

Table 3.13 - Total number of dogs examined with spinal injuries and spinal cord (n = 155)

Veterinary medicine clinics	2015	2016	2017	2018	Total animals
Veterinary centre "5th Element" in Vasylkiv	8	21	22	18	69
Private Veterinary Medicine Clinic "FOP Doletska K. V." in Kyiv	3	5	6	5	19
Private veterinary clinic "VetProfi" in Kyiv	7	8	6	10	31
Private clinic of veterinary medicine "Vet House" in Vinnytsia	2	11	10	13	36
Total animals	20	45	44	46	155

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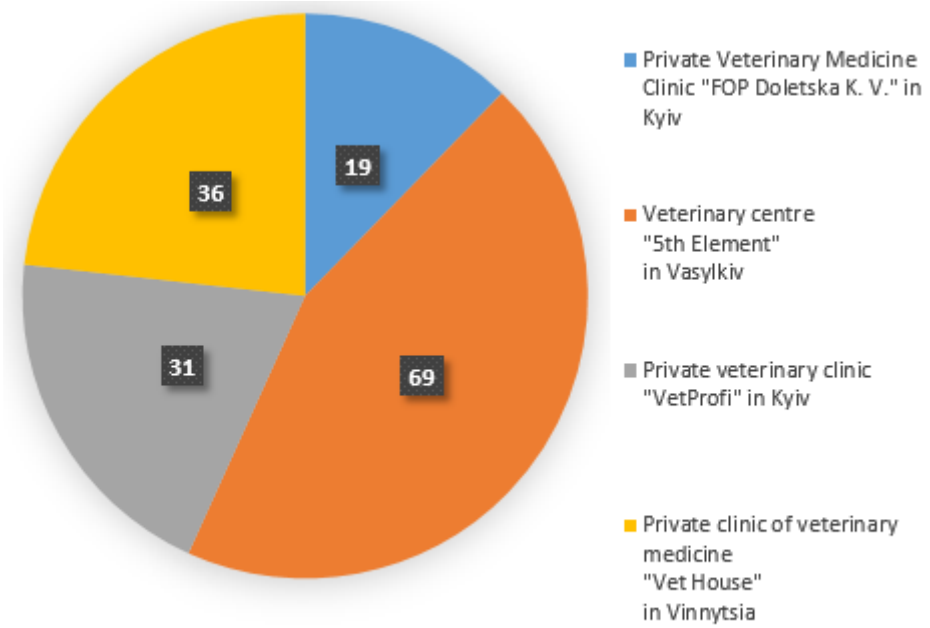

Fig. 3.8 - Quantitative proportion of examined dogs with diagnosed neurological disorders in veterinary medicine clinics in 2015-2018 (n = 155)

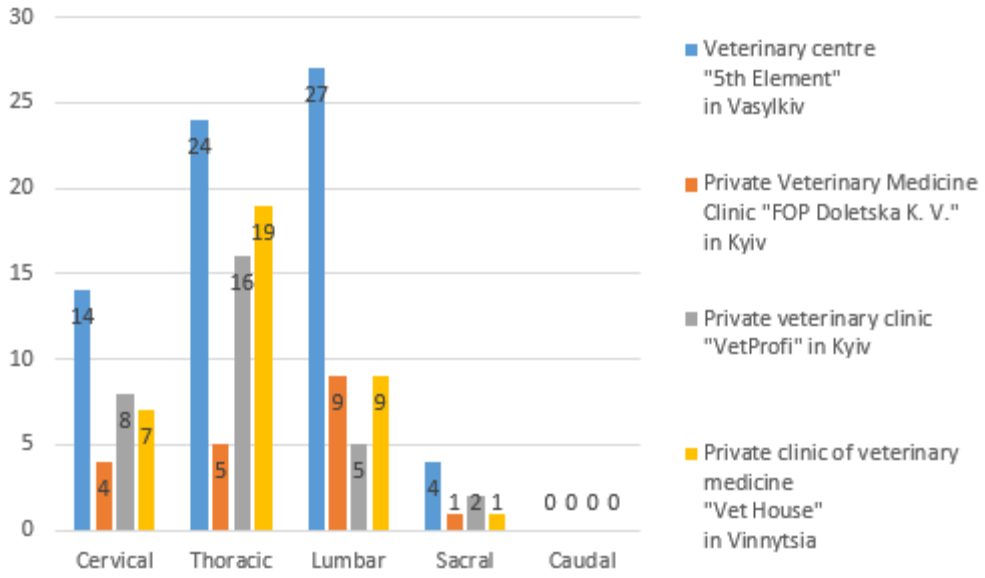

Fig. 3.9 - Total number of dogs examined with spinal cord and spinal cord injuries (n = 155)

Summarising the data obtained, the diagram shows the total number of spinal cord and spinal cord injuries in dogs (n = 155) by spinal column sections for the period 2015-2018 (Fig. 3.11).

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Table 3.14 - Number of cases of spinal cord and spinal cord injuries in different parts of the body

Veterinary medicine clinics	Number of cases of spinal injuries in different parts of the spine in dogs				
	Spinal regions				
	cervical (C)	thoracic (T)	Lumbar (L)	sacral (S)	caudal (Cd)
Veterinary centre "5th Element" in Vasylkiv	14	24	27	4	0
Private Veterinary Medicine Clinic "FOP Doletska K. V." in Kyiv	4	5	9	1	0
Private veterinary clinic "VetProfi" in Kyiv	8	16	5	2	0
Private clinic of veterinary medicine "Vet House" in Vinnytsia	7	19	9	1	0
Total animals n = 155	33 21.29 %	64 41.29 %	50 32.25 %	8 5.17 %	0

According to the localisation of the pathological process, spinal injuries were distributed as follows: in the cervical spine - 33 (21.29%), in the thoracic spine - 64 (41.29%), in the lumbar spine - 50 (32.25%) and in the sacral spine - 8 (5.17%).

Accordingly, the following spinal cord and spinal cord injuries were diagnosed in the cervical spine: Atlanto-axial subluxation, intervertebral hernia of Hansen type I and II, compression fracture of the vertebral bodies at the level of the third to sixth cervical vertebrae (C3 - C6), cervical spondyloarthrosis, syringomyelia (Table 3.15).

The following spinal cord and spinal cord injuries were diagnosed in the thoracic spine: intervertebral hernia of Hansen type I and II, compression fracture of the vertebral body at the level of the fourth thoracic - fifth thoracic vertebrae (T4 - T5), spondyloarthritis of the thoracic spine, myelitis, radicular syndrome, anterior spinal syndrome, spinal cord contusion, Schiff-Sherrington syndrome, and complete spinal cord rupture (Table 3.16).

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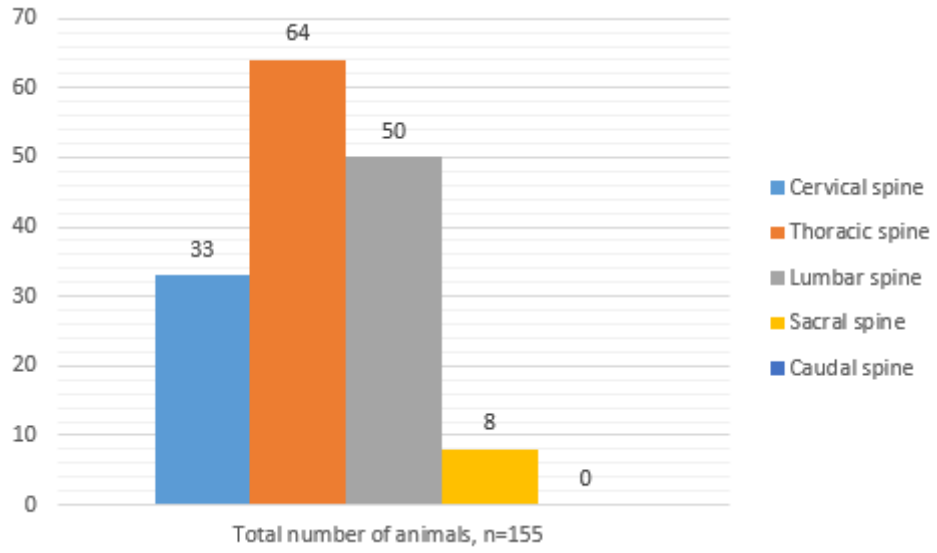

Fig. 3.10. Percentage of examined dogs with spinal cord and spinal cord injuries (n = 155)

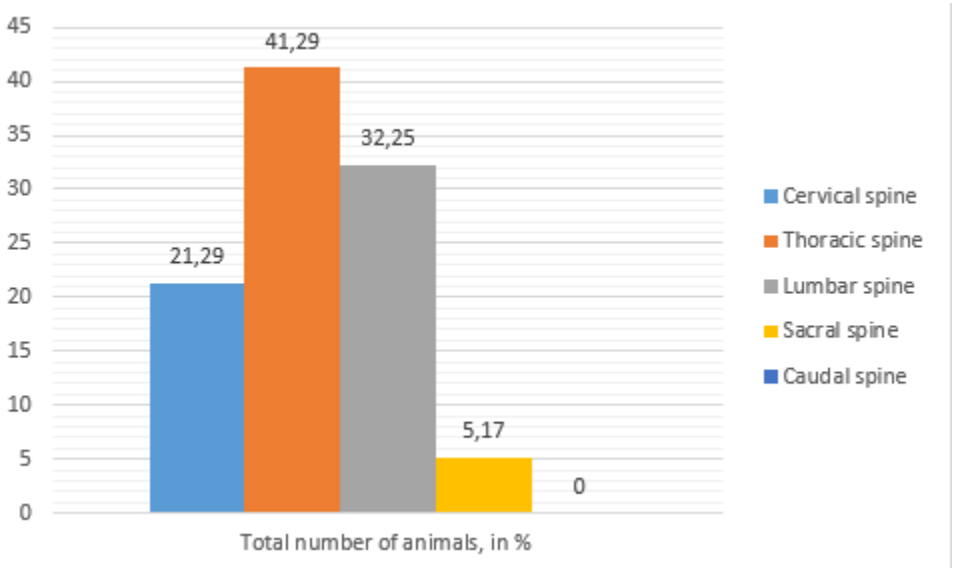

Fig. 3.11. Quantitative ratio of clinical cases of spinal injuries in dogs in 2015-2018 (n = 155)

Table 3.15 - Damage to the cervical spine and spinal cord

No. p/n	Damage to the cervical spine	Number of clinical cases
1	Atlanto-axial subluxation	9 (27.3 %)
2	Hansen type I intervertebral disc disease	7 (21.3 %)
3	Compression fracture of the C3 vertebral body	1 (3.0 %)
4	Compression fracture of the C6 vertebral body	1 (3.0 %)

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No. p/n	Damage to the cervical spine	Number of clinical cases
5	Cervical spondyloarthritis	3 (9.0 %)
6	Hansen type II intervertebral disc disease	11 (33.4 %)
7	Syringomyelia	1 (3.0 %)
Together		33

Table 3.16 - Damage to the thoracic spine and spinal cord

No. p/n	Damage to the thoracic spine	Number of clinical cases
1	Compression fracture of the vertebral body T4 - T5	1 (1.6 %)
2	Spondyloarthritis of the thoracic spine	3 (4.7 %)
3	Hansen type I intervertebral disc disease	15 (23.4 %)
4	Hansen type II intervertebral disc disease	13 (20.3 %)
5	Myelitis	2 (3.1 %)
6	Root syndrome	18 (28.1 %)
7	Anterior spinal syndrome	2 (3.1 %)
8	Schiff-Sherrington syndrome	5 (7.8 %)
9	Spinal cord contusion	4 (6.3 %)
10	Complete rupture of the spinal cord	1 (1.6 %)
Together		64

Table 3.17 - Damage to the lumbar spine and spinal cord

No. p/n	Damage to the lumbar spine	Number of clinical cases
1	Lumbosacral instability in the L7 region	1 (2.0 %)
2	Stenosis of the lumbosacral spine L7-S1 sacralisation of L7 - S1	5 (10.0 %) 1 (2.0 %)
3	Compression fracture of the L2 vertebral body	1 (2.0 %)
4	Compression fracture of the vertebral body L5 - L6	1 (2.0 %)
5	Compression fracture of the vertebral body L6 - L7	1 (2.0 %)
6	Cauda equina horse tail syndrome	10 (20.0 %)
7	Neoplasms in the area L6 - S1	1 (2.0 %)

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No. p/n	Damage to the lumbar spine	Number of clinical cases
8	Hansen type I intervertebral disc disease	2 (4.0 %)
9	Hansen type II intervertebral disc disease	3 (6.0 %)
10	Spinal cord contusion	2 (4.0 %)
11	Root syndrome	8 (16.0 %)
12	Anterior spinal syndrome	5 (10.0 %)
13	Spinal cord compression syndrome	2 (4.0 %)
14	Ascending syndrome	5 (10.0 %)
15	Discospondylitis	2 (4.0 %)
Together		50

The following spinal cord and spinal cord injuries were diagnosed in the lumbar spine: stenosis of the lumbosacral spine at the level of the seventh lumbar - first sacral vertebrae (L7 - S1), sacralisation of the vertebrae at the level of the seventh lumbar - first sacral vertebrae L7 - S1, cauda equina syndrome, intervertebral hernia according to Hansen type I and II, spinal cord compression syndrome, discospondylitis, lumbosacral instability in the area of the seventh lumbar vertebra (L7), radicular syndrome, compression fracture of the vertebral body at the level of the second lumbar vertebra (L2), anterior spinal syndrome, neoplasms in the area of the sixth lumbar - first sacral vertebrae (L6 - S1), spinal cord contusion, radicular syndrome, ascending syndrome (Table 3.17).

The following spinal injuries were diagnosed in the lumbar spine: vertebral body fracture at the level of the first sacral to the third sacral vertebrae (S1 - S3) (Table 3.18). No clinically significant injuries of the caudal spine were detected during the study period.

Table 3.18 - Damage to the sacral spine and spinal cord

No. p/n	Damage to the sacral spine	Number of clinical cases
1	Fracture of the vertebral body S1 - S2	3 (38.0 %)
2	Fracture of the vertebral body S2 - S3	5 (62.0 %)
Together		8

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General characteristics of spine and spinal cord diseases in dogs (n=155)

1. Atlanto-axial subluxation was diagnosed in 9 dogs aged 9 months to 4 years. The diagnosis was made in 3 Yorkshire Terriers aged 1 year, 1.4 years, and 2.3 years, 1 Chihuahua aged 3 years, 2 German Shepherds aged 2.5 years and 3.1 years, 1 Levretzky aged 6 years, and 2 Pekingese aged 3.3 years and 3.7 years. The main clinical signs at the time of initial admission consisted of a cautious gait when moving, severe pain when lifting the head up and turning it to the sides, accompanied by whining.

The degree of paresis of the thoracic and pelvic limbs was assessed by the Griffiths scale, as the progression of the process can result in tetraparesis with impaired urinary function. During the neurological examination, the degree of limb paresis was determined by clamping the base of the skin of the fingers with a Pean mosquito to determine the pull-off reflex. The main etiological factors for the occurrence of atlanto-axial subluxation are tearing or distortion of the ligaments between the atlas and the axial vertebrae, as well as fracture of the dentate process of the axial vertebra with simultaneous tearing of the ligaments. The vertebral dislocations were accompanied by traumatic damage to the IVD, rupture of its fibrous ring and varying degrees of spinal canal compression (Fig. 3.12). The absence of the dentate process of the axial vertebra and its dysplasia occurred in 46 % of observations, and ligamentous tear occurred in 24 % of cases. Compression of the basilar artery by the dentate process can lead to symptoms such as loss of spatial orientation, behavioural changes, and vestibular deficits.

The atlas and the axial vertebrae ensure the rotation of the skull. In this case, the first cervical vertebra (C1) rotates around the dentate process of the second cervical vertebra (C2). There is no intervertebral disc between the first and second cervical vertebrae (C1 - C2), so the interaction between these vertebrae is carried out by the ligamentous apparatus.

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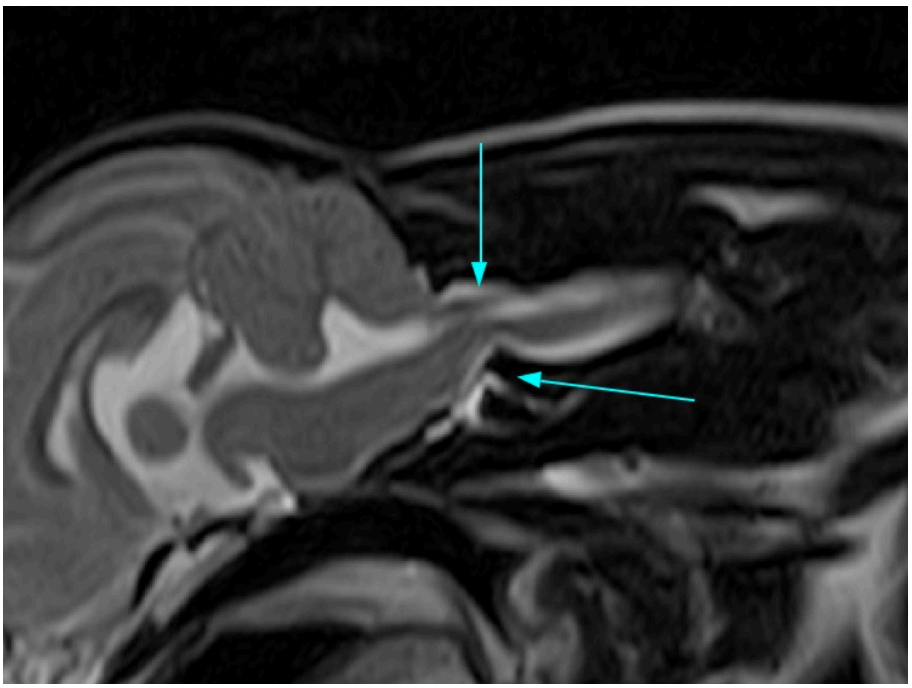

Fig. 3.12 - MR imaging of atlanto-axial instability in the sagittal plane in a 3-year-old Chihuahua dog named Nick. Intraoperative tooth separation of the axial vertebra was performed. Animal from n = 155

X-ray examination was performed in the lateral projection. Pillows were placed under the patients' heads to prevent excessive sideways rotation of the head. The distance between the dorsal arch of the atlas, which is small in normal conditions, increases by 2-3 times in the case of atlanto-axial subluxation. If an axial vertebral fracture could not be diagnosed by detection on frontal and sagittal tomograms, a "naso-occipital" image was taken through an open mouth in a supine position with the upper and lower jaw fixed. Atlanto-axial instability is well visualised on radiographs and this method of examination is quite sensitive.

Contrast myelography is contraindicated in atlanto-axial subluxation.

MRI: is effective in determining the structure of the spinal cord, but it is not very informative about bone tissue. This method can be an additional diagnostic tool for atlanto-axial subluxation.

A CT scan visualises the position of the axial vertebral tooth in the spinal canal well enough to assess its integrity. It is recommended to perform a "stress position" followed by bending the head at an angle of 45 degrees. CT semiotics includes an

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increase in the distance between the dorsal arch of the atlas and the crest of the axial vertebra, a violation of its degree of damage, and displacement of the epistrophic tooth into the spinal canal.

In cases of excessive pain, the dogs were given mild to moderate sedation (subsection 3.1.2).

2. We have diagnosed **intervertebral disc disease** in the cervical, thoracic, lumbar spine **according to Hansen type I** and **II** in 51 dogs aged 3 to 9 years of both chondrodystrophic and non-chondrodystrophic breeds.

Dysfunctions of the cervical spinal cord at the level of the UMN first cervical to fifth cervical vertebrae (C1 to C5) can cause neurological deficits in the four limbs. Clinically, this was manifested by ipsilateral hemiparesis or hemiplegia, pain in the thoracic and pelvic limbs, pain in the neck, mild spinal stiffness, and hypoventilation of the lungs. In some animals, involuntary urination and loss of pain sensitivity were observed, indicating partial spinal cord damage.

Hansen type I intervertebral disc disease (Intervertebral disc disease-1) was clinically manifested by acute pain and hemiparesis, partial loss of pain sensitivity. Diagnostics in this case was complicated due to the inability to perform proper fusion without the use of sedatives. Hansen type II intervertebral disc disease (Intervertebral disc disease-2) was most often chronic, and in case of progression of degenerative changes, spinal injuries, excessive rotations during jumping, it causes a rupture of the fibrous ring with the development of root syndrome. The complexity of the course of intervertebral disc disease depends on the location and compression of a pair of spinal nerves.

A disc bulge with herniation can occur on one side of the dorsal longitudinal ligament that forms the bottom of the spinal canal. As a result, one of the limbs is more affected than the other, which was observed in most affected dogs. Irritation of the nerve endings is manifested by impaired support on the affected limb in a standing position and significant pain, which causes lameness.

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Table 3.19 - Degrees of neurological disorders

Degree	Neurological disorders	Total number of animals
0	Normal, no pain syndrome	19
I	Only pain syndrome (hyperesthesia)	12
II	Impairment of proprioception only (proprioceptive ataxia) or ambulatory paraparesis, limb resistance function is preserved	6
III	Non-ambulatory paraparesis, impaired resistance function of the limb	2
IV	Non-ambulatory paraparesis, impaired limb resistance function, urinary disorders (deep pain sensitivity)	7
V	Non-ambulatory paraparesis with no deep pain sensitivity	5

The degree of narrowing of the spinal canal is directly proportional to the degree of spinal cord compression and affects the manifestation of neurological disorders. In a group of 51 dogs, spinal cord compression was found to varying degrees. The degree of spinal cord compression corresponds to the nature of the neurological deficit (Table 3.19).

The interpretation of X-rays revealed a narrowing of the intervertebral space between the articular processes, mineralisation of the pulp nucleus in the spinal canal, and an increase in the radiological density of the intervertebral foramen. In the case of intervertebral disc disease according to Hansen type II, the diagnosis was difficult, while in the presence of intervertebral hernia type I, the diagnosis rate was up to 80%. Indirect signs indicating IVDD-I include sclerosis of the laminae in the form of spondylosis and osteophyte formation, mineralisation of the intervertebral disc, a "vacuum phenomenon" in the nucleus pulposus and a decrease in the distance between the laminae (Fig. 3.13).

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Myelography, as a method of visual diagnostics, helped to detect an increase in the ventral column in the area of intervertebral disc rupture with thickening of the dorsal column in the same area. In type I IVD disease, part of the nucleus pulposus substance is displaced dorsally, in particular, the dorsal column may be displaced ventrally. If a block of contrast columns is detected over several vertebrae, this indicates spinal cord edema with underlying IVD disease or spinal column injury.

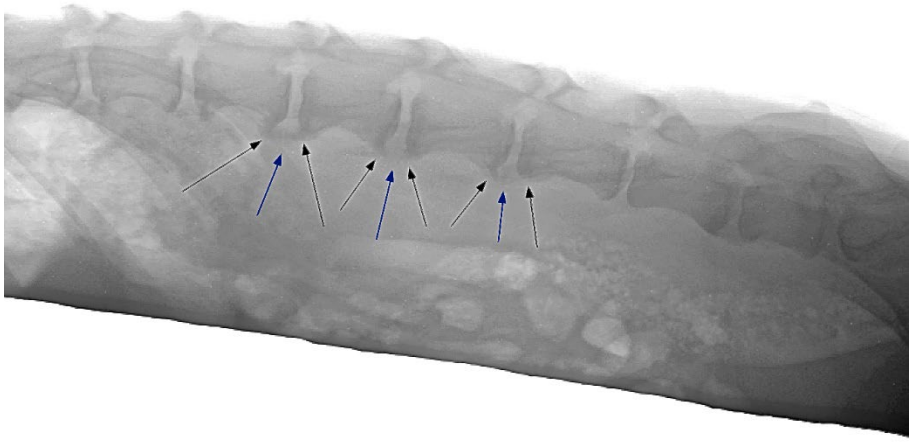

Fig. 3.13. An X-ray of the lumbar spine of an 11-year-old German Shepherd dog named Phil shows spondylosis at the level of the second to fifth lumbar vertebrae (L2 - L5) - "black arrows". In the segment L2 - L5 of the intervertebral spaces, indirect signs of intervertebral disc protrusion are visualised - "blue arrows". Animal from n = 155

In doubtful cases, an additional MRI diagnosis in T2-weighted mode was prescribed. The obtained results of the studies showed narrowing of the cerebrospinal space in the areas of intervertebral disc displacement and detection of detrital material above the border of the intervertebral foramen. The main signs of type I intervertebral disc disease were the detection of foreign mineralised substance in the spinal canal. The spinal cord was also found to be displaced in the opposite direction from the newly formed volume, foreign mineralised substances at the level of the intervertebral disc with a characteristic "blurred" perimeter contour. To date, myelography is the main method for diagnosing IVD hernia, with a diagnostic value of 90%. In relation to Hansen type II intervertebral disc disease, protrusions are often not detected. The

examination requires proper animal positioning to prevent questionable results.

CT myelography allows detecting changes in the contours, structure and radiographic density of the contrast ring in the area of a herniated disc or their complete visualisation. In the diagnosis of Hansen type II intervertebral disc disease, CT myelography contributed to a detailed assessment of the compression effects of protrusion with 99% sensitivity.

The main important feature of CT scan (type I intervertebral disc disease) is determination of the density of the soft part of the fibrous ring structure. Its density is close to that of the spinal cord (80-100 HU). The contour of the soft tissue of the fibrous ring is noted on 3 sections that have passed along the level of the intervertebral disc. The "vacuum" phenomenon is detected at the level of the disc nucleus pulposus substance. In the diagnosis of intervertebral disc disease according to Hansen type II, it is almost impossible to diagnose only by assessing the compression function of a single protrusion, since it looks like a "heterogeneous mass". It has been shown that during the study period there was no clear correlation between the occurrence of Hansen type I and II intervertebral disc herniation in dogs and the age criterion. Accordingly, young dogs have a Hansen type II herniated disc, while dogs of the older group over 7 years of age have a Hansen type I herniated disc.

The main signs of intervertebral disc degeneration are: changes in the height and nature of the signal on a T2-weighted image; changes in disc contours; fibrous ring tears; changes in the lamina propria.

3. Compression fractures of the vertebral body based on comprehensive examinations in 14 dogs were diagnosed at the level of the third to sixth cervical vertebrae (C3 - C6), the fourth to fifth thoracic vertebrae (T4 - T5), second lumbar vertebra (L2), fifth - sixth lumbar vertebrae (L5 - L6), sixth - seventh lumbar vertebrae (L6 - L7), first - third sacral vertebrae (S1 - S3). Fractures of the cervical spine were diagnosed in 2 cases, 1 in the thoracic spine, 3 in the lumbar spine, and in the sacral spine in 8 dogs.

Paraplegia was observed in the thoracic spine with vertebral body fractures, and at the level of the third thoracic - third lumbar vertebrae (T3 - L3), Schiff-Sherrington

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syndrome with clinical signs of hyperextension of the thoracic extremities could sometimes occur.

When using the radiological method, the animals were placed in the lateral and dorsal projection. Depending on the location of the fracture, the vertebrae acquired a wedge-shaped shape, its length along the ventral contour was reduced compared to the neighbouring vertebrae. The vertebral length could be reduced by ¼ and 1/3, which caused excessive compression of the intervertebral disc. At the same time, the spinal axis was deformed at an angle, the apex of which was directed dorsally and in the area of the injured vertebra. Special attention was paid to the change in the shape and constancy of the width of the spinal canal with vertebral fragments, shortening of the vertebral bodies with compaction of the spongy tissue, and narrowing of the intervertebral gap.

Radiological and CT semiotics: displacement of vertebral segments, interruption of the bottom line or dorsal wall of the spinal canal, narrowing of the intervertebral disc space, shortened vertebral segments. The radiological method of diagnosing vertebral fractures is the main one.

MRI: bone tissue damage; soft tissue damage; determination of the state of the ligamentous complex (yellow ligament, supracondylar ligament).

4. Syringomyelia was diagnosed by tomography in 1 Belgian Griffin dog aged 7 months. For 3 weeks, the animal had sensory ataxia, loss of balance when walking, limb tremors, and sometimes inspiratory dyspnoea. The main sign is the symptom of "combing the air", reminiscent of removing a collar, which may not be on the dog.Radiological diagnostics do not allow for a definitive diagnosis, but it is possible to visualise spinal stenosis. The use of MRI to confirm the diagnosis of syringomyelia is crucial because it is necessary to carefully examine the brain, cerebellar size and soft tissue in relation to the atlas. The disease of an animal at an early age indicates improper breeding and such dogs are not allowed to be bred further.

MRI is the best method for diagnosing syringomyelia in dogs.

5. 6 dogs were diagnosed with **cervical** and **thoracic spondyloarthrosis** by radiology**.** The disease was recorded in 1 Pekingese aged 11 months, 1 Toy Terrier

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aged 1.3 years, 2 Yorkshire Terriers aged 1.4 years and 1.9 years, respectively, 1 French Bulldog aged 2 years and 1 German Shepherd aged 11 years. The symptoms of the disease were quite pronounced and manifested as sensory ataxia of the thoracic limbs, less often on the pelvic limbs. During fast movement, it was almost not manifested by limb entanglement, while when the animal was turned sideways, the dog lost its balance and lay down on its stomach. During the neurological examination, the proprioception of the limbs was impaired, and in some patients, claw abrasion was noted, indicating shuffling of the limbs during walking.

X-ray examination reveals hyperplasia of the dorsal fibrous ring of the intervertebral disc and deformation of the vertebral bodies with exostoses.

Myelography: is the main method of diagnosing spondyloarthrosis of the cervical and thoracic spine**.**

6. Myelitis was diagnosed by clinical, neurological, X-ray, cytological and biochemical examination of cerebrospinal fluid in 2 dogs: a short-haired dachshund aged 4 years and a Pekingese aged 3.7 years.

Traumatic myelitis occurred as a complication of spinal fractures, also due to displacement of intervertebral discs.

After the neurological and X-ray examination and a full course of conservative treatment in dogs, it was concluded that restoration of spinal cord function is possible in animals with II and III degrees of neurological disorders. In dogs with grade IV, there is a low probability of restoring motor function. In dogs with the V degree of neurological impairment and lumbar spinal cord injury syndrome (plegia), there is virtually no possibility of restoring spinal cord function.

7. During the neurological examination, 26 animals were diagnosed with **radicular syndrome**. The radicular syndrome is manifested by a large number of symptoms caused by spinal nerve compression. It is characterised by a pronounced painful effect in the part of the spine where the spinal roots are compressed. Nerve compression in the area of the sixth cervical vertebrae - second thoracic vertebrae (C6 - T2) or the fourth lumbar vertebrae - second sacral vertebrae (L4 - S2) with lateral disc displacement is characterised by severe pain in the lumbar region (Fig. 3.14).

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Intervertebral disc herniation can occur on the side of the dorsal longitudinal ligament, which forms the bottom of the spinal canal. As a result, one of the limbs is more affected than the other. Excessive irritation of the nerve endings is manifested by impaired support on the affected limb in a standing position and significant pain, which causes lameness during movement.

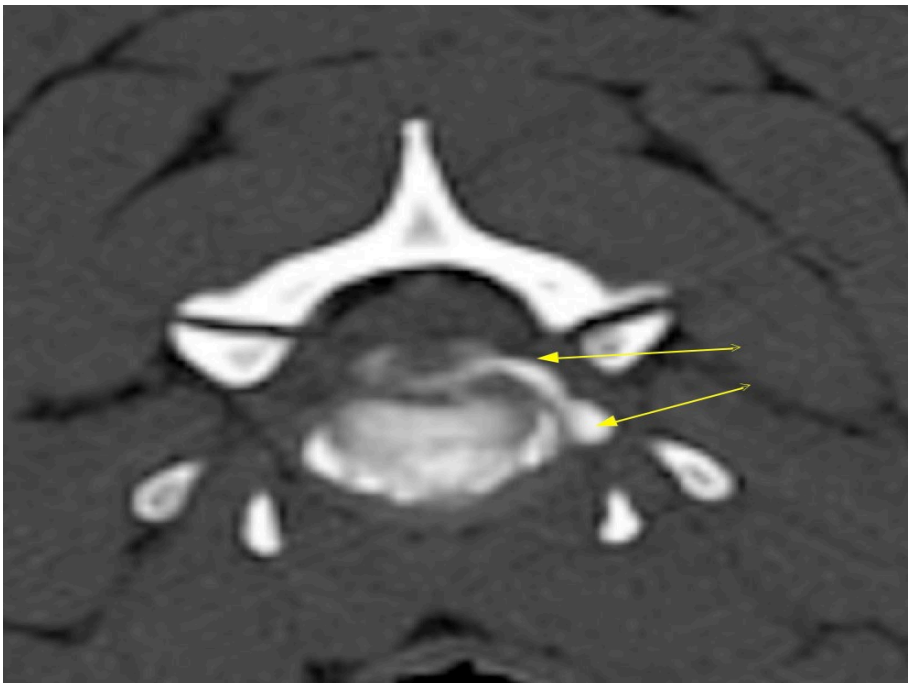

Fig. 3.14 - MR imaging with yellow arrows shows the nerve root with clear contrast staining, indicating compression from the fibrous ring and leakage of the nucleus pulposus. Clinically, the patient has a severe pain syndrome. Dachshund, 4 years old, ♂, Phil (E2).

Diagnosis is possible only with MRI to determine the location of nerve root compression in the segmental plane.

8. Anterior spinal syndrome was diagnosed in 7 dogs based on the results of neurological examination and tomographic examination: 4 short-haired dachshunds aged 3.3, 4.0, 4.3 and 5.2 years, respectively, 1 Pekingese aged 6.1 years, 1 Central Asian shepherd dog aged 3 years, 1 pug aged 3.5 years.

Anterior spinal syndrome is quite often diagnosed in spinal cord injuries. It is characterised by incomplete damage and occurs in vertebral compression fractures, sometimes with dislocations, and IVD herniations. The syndrome occurs with

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widespread bilateral spinal cord injury. Below the level of injury, motor, sensory and autonomic disorders develop. Only vibration and proprioceptive sensitivity remain intact. The syndrome often develops as a result of compression of the anterior spinal artery in the spinal canal.

Diagnosis of the syndrome is possible only with the use of MRI to determine the location of spinal cord compression in the segmental plane.

9. After spinal cord injuries, 5 dogs were diagnosed with **Schiff-Sherrington** syndrome: 2 German shepherds aged 5 and 6 years, 2 short-haired dachshunds aged 5 and 6 years, and 1 French bulldog aged 8 years.

In some cases, Schiff-Sherrington syndrome is detected in dogs when the spinal cord is severed between the second thoracic and fourth lumbar vertebrae (T2 - L4) and tends to develop clinical signs within 24 to 48 hours. Using the functioning peripheral muscles, the animal is able to rise and attempt to walk; these are involuntary movements, but they can be mistaken for voluntary movements. The syndrome is observed in severe spinal cord injuries in the thoracolumbar spine. The spinal reflexes are correct, and the sensation of the thoracic limbs and voluntary motor function are normal. A neurological examination reveals muscle atrophy, which means that the descending conduction pathways are involved in the pathological process. After a serious spinal cord injury of the thoracic spine with trauma to the ascending inhibitory pathways, the tone of the extensor muscle of the thoracic limb can be significantly increased.

During the clinical examination of the dogs, the following clinical signs were noted: hyperextension of the thoracic limbs, severe paresis of the pelvic limbs. In 2 cases, significant spinal stiffness was detected, and in 2 cases, involuntary urination was noted. Respiratory and cardiovascular system: tachycardia, tachypnea; consciousness was present, signs of dementia were not identified. Periodically, swimming movements were observed with a painful syndrome.

Radiography: is the main method of instrumental diagnostics of the nature and extent of vertebral damage in the acute period of spinal trauma. It helps to collect information about the condition of the spine and spinal cord in a short period of time.

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To determine the level of damage to the spine, preference is given to radiographs taken in the lateral projection.

MRI: the examination is performed in the thoracolumbar spine to visualise damage to the ligamentous apparatus and intervertebral discs, as well as spinal cord tears.

In cases of excessive pain, the dogs were given mild to moderate sedation (subsection 3.1.2).

10. Spinal cord contusion was diagnosed in 6 dogs: 2 short-haired dachshunds aged 2 and 4 years, 2 French bulldogs aged 4 and 4.4 years, 2 German shepherds aged 5 and 7.7 years.

In chondrodystrophic breeds, the diameter of the spinal cord in relation to the diameter of the spinal canal is proportionally much larger than in non-chondrodystrophic breeds. The size of contrast lines is smaller in the latter.

X-ray: allows to detect concomitant fractures and subluxations of vertebrae.

CT scan: contusion is manifested by a local decrease in spinal cord density, with haematomas showing a hyperdense area with a density of 50-90 HU.

MRI: T1-weighted mode is recommended. Acute period: SC edema. MR signal is hypointense compared to intact areas. SC interruption: in the sagittal plane. Chronic period: multisegmental gliosis of the SM looks like a hyperintense zone.

11. A complete spinal cord rupture was diagnosed in 1 dog of the short-haired dachshund breed aged 5.2 years.

Complete rupture of the spinal cord in closed spinal fractures is quite rare. Spinal cord dislocation is caused by injuries that are accompanied by dislocation of more than 1/3 of the vertebral body. Acute cerebral oedema with insufficient blood supply occurs both as a result of trauma and as a result of the progression of edema. As a result, vascular innervation is impaired, leading to stasis, haemorrhage, and, in case of prolonged blood stasis, necrosis of nervous tissue.

While X-rays and CT scans allow to assess changes in the vertebral bone tissue, MRI easily visualises damage to the ligamentous apparatus and intervertebral discs and additionally confirms the suspicion of a partial or complete spinal cord tear.

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12. Lumbosacral instability in the area of the seventh lumbar vertebra (L7) was diagnosed in 1 German Shepherd aged 6 years, ♀, weighing 37 kg.

Below is a fragment from the neurological protocol of a sick animal.

Examination of the head: mental status - the animal is conscious, no convulsions or uncoordinated movements were observed. Examination of cranial nerves: no abnormalities were found. Gait assessment: paresis of the pelvic limbs, forced turns of the trunk as a result of pain. Examination of the thoracic and pelvic limbs: jumping is impossible, conscious proprioception is present. Spinal reflexes: no abnormalities were detected during the examination of the thoracic limbs. Paresis was noted in the pelvic limbs. Anal sphincter tone was reduced, spontaneous faecal excretion was noted. The tail position is forced, slightly bending towards the pelvic limbs. Less frequently, involuntary uncontrolled urination is observed, and voluntary tail movements are limited. No atrophy of the pelvic limbs muscles was detected at the first stage of the examination. Pain sensitivity: + 1 point - hyperesthesia.

Injury location: lumbosacral spine. Severity of injury: moderate.

Study of the gradation of ASIA motor function: 1 - 2 points. The motor function of the extremities was examined and it was found that there are slight muscle contractions that can be determined visually or palpably. Sometimes there are movements in the direction of gravity. The number of points obtained indicates the depth of the injury and impaired nerve impulse conduction.

ASIA sensory function grading study: + 1 point - hyperesthesia. Slight sensitivity to needle tingling of the skin and muscles in the corresponding somatomas. ASIA impairment severity scale: C (incomplete). Motor function below the neurological level is preserved, but more than half of the key muscles below the neurological level have a strength of less than 3 points.

The X-ray examination revealed lumbosacral instability due to displacement of the sacral vertebra (S1) relative to the body of the seventh lumbar vertebra (L7), which leads to excessive instability in this area and is associated with underdevelopment of the articular process.

13. Stenosis of the lumbosacral spine at the level of the seventh lumbar - first

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sacral vertebrae (L7 - S1) was found in 6 dogs: 2 Central Asian shepherds aged 4 and 5 years, 1 Mastino Neapolitano aged 4 years, 2 pugs aged 5 and 7 years, 1 French bulldog aged 3 years.

Degenerative changes in the lumbosacral spine cause pain in the area of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1) in German Shepherds, German Shepherd mixes, and relatively less often in small breeds. These changes include proliferation of the interosseous ligament, formation of osteophytes on the vertebral surfaces, degenerative disease of type II IVD and deforming spondylosis in the area of the seventh lumbar vertebrae - the first sacral vertebrae (L7 - S1). This leads to the development of spinal canal stenosis and compression of nerve roots in the area of the seventh lumbar vertebrae - the fifth caudal vertebrae (L7 - Coss 5).

One of the etiological factors in the development of the pathology is vertebral malformations, which are most common in the thoracic and lumbar spine of dogs. These include congenital malformation of half a vertebra, split vertebrae in the form of a butterfly, transitional vertebrae and vertebral block.

Compression of the Cauda equina or individual nerve roots in the area of the sixth lumbar vertebrae - the first sacral vertebrae (L6 - S1) as a result of narrowing of the spinal canal or the presence of spondylolisthesis, accompanied by secondary reactions in the ligaments, disc apparatus or vertebral bodies. Due to the loss of function, primarily of the sciatic, pudendal and caudal nerves, sensory, motor or autonomic disorders occur.

On the basis of plain X-rays, stenosis of the spinal canal, ventral displacement of the first sacral vertebra (S1) relative to the seventh lumbar vertebra (L7), and changes in bone tissue are noted. It is also recommended to perform stress X-rays and contrast myelography to exclude neoplasms, spondylitis, arthrosis, prostatitis, etc.

Abnormally developed vertebrae: well visualised on X-rays.

Myelography can often be uninformative due to spinal canal stenosis and insufficient filling of the CSF with contrast medium.

Abnormally developed vertebrae: myelography provides an opportunity to assess compression on the vertebral side.

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MRI is the recommended method for diagnosing lumbosacral stenosis compared to CT myelography.

Abnormally developed vertebrae: low level of bone tissue visualisation. MRI diagnostics is performed to establish spinal cord compression, which can cause neurological deficits.

CT scan allows for good visualisation of the intervertebral disc structures displaced into the spinal canal. The combination of CT with myelography is highly informative. The disadvantage of the method may be severe stenosis of the spinal canal.

Abnormally developed vertebrae: has high sensitivity to the structure of such vertebrae and allows timely determination of compression function due to the established diameters of the spinal canal of abnormally developed vertebrae and the diameter of the spinal cord. The diagnosis makes it possible to identify a trapezoidal shape in the middle sagittal plane, vertebral body splitting, and narrowing of the spinal canal in the area of the arch of the abnormally developed vertebra.

CT myelography: often has no advantages over the standard CT method, so it is practical to use MRI for the differential diagnosis of spinal cord diseases.

14. One of the important neurological disorders that was found in 10 animals is the **"horse tail" syndrome** or Cauda equina.

The main causes of the syndrome are discospondylitis, discitis, neoplasms on the vertebrae and genetic disorders of the spine.

The neurological disorder often causes changes in the behaviour of dogs, which leads to forced inactivity due to severe pain. The initial period of the syndrome is characterised by asymmetry of the symptoms of the injury. In some cases, Kernig's symptom and protective reflexes of the pelvic limbs are noted. The syndrome is caused by injuries of the lumbar and sacral vertebrae, which are accompanied by haemorrhages in the sub- or epidural spaces. Sometimes, a neoplasm in this area or a prolapsed IVD causes the clinical picture of the syndrome.

The cauda equina syndrome is accompanied by peripheral paralysis or paresis of the distal pelvic limbs, dysuria, and anaesthesia in the pelvic and perineal regions. During the clinical examination, a characteristic severe radicular pain in the lumbar

region is noted. Conductive sensory disturbances often increase from top to bottom. Over time, dogs experience a sharp decrease in body weight, even muscle contracture, and in the later stages, the absence of Achilles reflexes, sensory disturbances in the area of innervation of the damaged roots, and vasomotor trophic disorders of the pelvic limbs. Nerve damage caused by nerve root compression can be aggravated by ischaemia caused by blood vessel compression.

In cases of excessive pain, the dogs were given mild to moderate sedation (subsection 3.1.2).

15. Neoplasms in the L6 - S1 region are rarely observed in the LSR, so they may go unnoticed in the vast majority of patients. Sometimes, a neoplasm in the sixth lumbar vertebrae - first sacral vertebrae (L6 - S1) in combination with the prolapse of the IVD causes the clinical picture of Cauda equina syndrome or lumbosacral stenosis.

It should be noted that these tumours do not metastasise to vital organs such as the lungs, liver and brain. By compressing the spinal cord, they cause paralysis of the pelvic or thoracic and pelvic limbs, and lead to urinary and defecation disorders. The neurological symptom complex manifested in dogs of all ages can be caused by neoplasms on the vertebrae.

X-ray diagnostics: tumours are often invisible due to their soft tissue structure. In rare cases, it is possible to visualise a significant tumour size.

MRI: this method is considered to be more sensitive to vertebral neoplasms than the use of contrast agents.

MRI in case of metastases: iso-hypointense signal on T1-weighted imaging; hyperintense signal in the sequence of suppression of MR signal from adipose tissue (STIR); marked accumulation of contrast agent.

CT: has a very high efficiency in terms of visualisation of neoplasms with subsequent injection of contrast medium. CT semiotics: low-density areas of lytic destruction, good contrast agent accumulation, presence of soft tissue component.

The CT scan depends on the degree of mineralisation of the metastasis, if they are visualised. Lytic metastases appear as areas of destruction with indistinct irregular contours. Osteolytic metastases often grow into the spinal canal, root canals, and

paravertebral tissue. Sclerotic lesions are defined as hyperdense, with irregular contours that do not extend beyond the vertebrae.

Contrast myelography: indicated in cases where it is necessary to determine the site of compression with subsequent MRI.

The tomographic method determines the presence of a tumour on the vertebral bodies with high confidence, noting the risk of metastasis to deep tissue layers. The diagnostics revealed the localisation of the tumour in the area of the sixth lumbar vertebrae - the first sacral vertebrae (L6 - S1) in 1 French bulldog aged 6 years with neurological deficit and Cauda equina syndrome.

16. Spinal cord compression syndrome was detected in 2 dogs of the short-haired dachshund breed aged 4 and 5 years.

Spinal cord compression is classified according to the time of development as:

a) acute, which occurs at the time of injury; b) early, which occurs a day after the injury; c) late, which occurs several days later.

According to the location, spinal cord compression is classified as:

a) posterior - caused by the development of a haematoma; b) anterior - as a result of the interaction of the body of a broken and displaced vertebra or intervertebral disc; c) internal - caused by an intracerebral haematoma resulting from a spinal cord injury, swelling of the spinal cord and its membranes.

Anterior spinal cord compression syndrome is often caused by a fracture of the cervical vertebrae with partial displacement, the occurrence of a Hansen type I herniated disc with subsequent compression of the anterior artery. According to the degree of spinal cord compression, we distinguish between: a) complete, accompanied by a complete impairment of functional conduction; b) partial, with some nerve conduction preserved.

Radiographically, the causes of spinal cord compression syndrome cannot be detected, except when the nucleus pulposus of the intervertebral disc penetrates deeply into the spinal canal. Also, epidural haematomas and acute spinal cord edema are not visualised. Contrast myelography followed by radiography is appropriate. In this case, some spinal cord injuries can be diagnosed in a timely manner. The reason for poor

visualisation of the level of the damaged intervertebral disc on myelograms is spinal cord edema, which blocks the contrast column over several vertebrae and several levels of intervertebral discs, respectively. As a result, there are grounds to characterise myelography as a sensitive method for determining the location of spinal cord compression, but it does not allow determining the etiological factors that caused the injury.

If the comprehensive diagnostics did not provide reliable diagnosis, MRI examination is performed.

In cases of excessive pain, the dogs were given mild to moderate sedation (subsection 3.1.2).

17. Acute spinal cord injuries causing secondary complications contributed to the development of **ascending syndrome** in 5 dogs, 2 of which were German shepherds aged 6 and 7 years, 1 short-haired dachshund aged 4 years, 1 Rottweiler aged 7 years, and 1 French bulldog aged 4 years.

The main etiological factors that cause the development of degenerative myelopathy are classified according to the following criteria: 1. traumatic nature (fracture, subluxation, dislocation of vertebrae). 2. Diseases of intervertebral discs according to Hansen type I. 3. Vascular pathologies: fibrocartilage ring embolism (acute and painless). One of the causes of spinal cord compression may be a herniated disc according to Hansen type I. Ascending myelomalacia is the destruction of spinal cord tissue during its upward softening, destroying the parenchyma and roots of the spinal nerves that pass to the diaphragm and intercostal muscles. The herniated nucleus pulposus of the intervertebral disc due to a rupture of the fibrous ring causes rapidly developing spinal cord ischaemia, which is associated with damage to the spinal cord tissue.

To establish the diagnosis, it is recommended to use a magnetic resonance imaging scanner, as visualisation of the spinal cord by radiography without the introduction of contrast agents is impossible. Given the danger of developing ascending syndrome after spinal surgery, its use in practice is advisable. Not only individual anatomical structures (cerebrospinal fluid spaces, spinal cord, cerebellum) are better

displayed on MR imaging of the NS, but grey and white matter of the brain, the spinal cord itself, areas of edema, haemorrhages, cysts and neoplasms are clearly distinguished. As a result, we assess the structure of the spinal cord, its integrity, the nature of compression, and changes in the surrounding tissues. This has many advantages in relation to myelopathies.

The use of a contrast agent in myelography allows to identify changes in meningeal structures when the blood-brain barrier becomes permeable. Normally, the contrast agent around the spinal cord appears on radiographs as two parallel bands, ventral and dorsal columns. Without contrast medium, the contour of the spinal cord is not defined, only the contours of the vertebral bone tissue are visualised. When myelomalacia develops, the contrast agent can mix with particles of necrotic tissue of the damaged area of the spinal cord, which also leads to the appearance of fluid turbidity or heterogeneity. If the disease is not acute, myelography indicates a physiological norm or local atrophy of the spinal cord. When a contrast agent is injected into the CSF, we can observe its entry into the circulatory system.

CT scan: spinal cord density is 36 HU (normal 24 - 44 HU). Reduced density in the foramen foramen and periphery of the spinal cord is normal due to the presence of epidural fat. The CT scan shows a very dilated central canal.

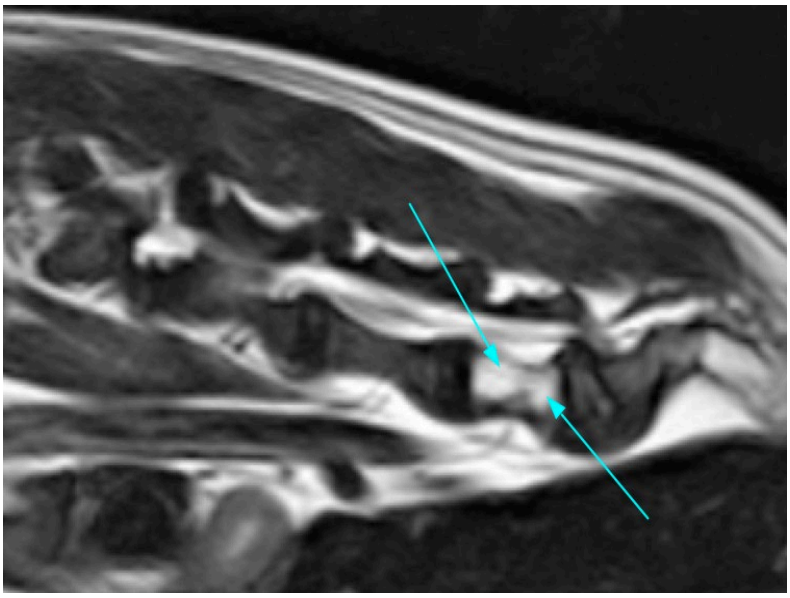

Fig. 3.15 - MR imaging with post-traumatic lumbar spinal discospondylitis in an 8-year-old Pekingese, Simon. Sagittal plane. Animal from n = 155

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18. After a thorough orthopaedic and neurological examination, 2 animals were diagnosed with severe pain in the lumbosacral spine according to the Griffiths scale. According to the results of X-ray examination, 2 Labrador dogs aged 7 years and a Pekingese aged 8 years were diagnosed with lumbar **spinal discospondylitis** (Fig. 3.15). Clinical signs were manifested by spinal pain, inactivity, and severe spinal stiffness.

The symptom complex of the pain syndrome was manifested by signs of forced body position in space with pelvic tremors and weakness of the thoracic limbs. After prolonged lying down with subsequent rising for 10-15 minutes, whining and severe anxiety were noted. Since discospondylitis is acute, it can contribute to the development of secondary complications in the form of spondyloarthrosis.

Radiographically, discospondylitis is diagnosed by signs of vertebral deformity with loss of their contours, displacement of the ventral processes of the vertebral bodies from the intervertebral spaces, and narrowing of the MP. The diagnostic method is quite sensitive and informative to changes in the density of the closure plates. Formed osteophytes in the spinal canal are relatively poorly detected.

X-ray contrast myelography identifies the area of possible compression, but does not improve the visualisation of bone tissue.

MRI: not a sensitive method for mineralised formations, but informative for concomitant complications, such as Hansen type II herniated discs. It is an informative diagnostic method in relation to spinal cord compression.

CT myelography: used to diagnose secondary complications in the form of Hansen type II intervertebral disc disease, protrusion, etc.

CT is a very sensitive method for detecting minor changes in the laminae and osteophytes of different sizes. The early diagnosis of discospondylitis and spondyloarthritis is based on this method and is considered optimal. The main signs on a CT scan are the presence of mineralised fragments on the lateral and ventral sides of the vertebral body. In some cases, there is a border between the vertebral bodies and mineralised fragments that unite 2 or more vertebrae. Depending on the stage of the process, the density of the fragments can vary from normal to increased.

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From the above number of dogs with spinal cord and spinal cord injuries (n = 155), as evidenced by the data in Tables 3.15-3.18, and a thorough orthopaedic and neurological examination, 3 experimental groups of animals (E1, E2, E3) were formed, with n = 5 in each group. The groups included dogs that were regularly monitored for their clinical condition, for the necessary blood and CSF sampling and instrumental research methods, which amounted to 9.7% (15 dogs). The control group consisted of clinically healthy animals, n = 5. No treatment was performed.

2.3. Testing of methods of conservative treatment of animals with neurological syndromes in acute spinal injuries

Conservative treatment of animals with SCI was aimed at reducing pain and preventing the development of shock, improving blood microcirculation in areas of spinal cord injury and stimulating recovery processes. The clinical effectiveness of conservative treatment was determined by providing care to dogs in a veterinary clinic and hospital for two years and six months. Each group included animals with the same neurological disorders, including radiological and myelographic changes. In particular, the absence of complications, age, sex and breed were taken into account.

Treatment was started in the acute period of the disease, which included the first 5 days after the spine and spinal cord injury with the subsequent development of spinal shock. In the majority of patients, the main etiological factor is neuronal damage due to spinal cord compression. The tomographic diagnostic method is particularly valuable, as it provides detailed visualisation of bone and soft tissue, including nerve roots, determines the anatomical integrity of the spinal cord and identifies the development of ascending myelomalacia, which is a life-threatening condition.

The clinical trial period lasted from 2 to 14 months. The animals of the control and experimental groups were kept in the same inpatient clinic conditions with the same feeding and care requirements.

The composition of the control (C) and experimental groups (E1, E2, E3), clinical forms of damage to the thoracolumbar spine and spinal cord, and means of

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medical treatment are shown in Table 3.20.

Table 3.20 - Formation of control and experimental groups according to neurological symptoms with the definition of a set of drugs

Animal groups, their composition and diagnosis	Number of animals	A complex of drugs
Control (C) 1. Cauda equina syndrome (horse's tail) 2. Root syndrome	5	1. Analgin 50 % (500 mg/ml) 2. Dexa-kel 2 % (20 mg/ml)
First experimental (E1) 1. Cauda equina syndrome (horse's tail) 2. Lumbosacral syndrome 2.	5	1. Depo-Medrol (40 mg/ml) 2. Vitaxon 3. Armadin (50 mg/ml)
Second experimental (E2) 1. Spinal cord compression syndrome caused by intervertebral disc disease according to Hansen And like. 2. Root syndrome	5	1. Prednisolone (30 mg/ml) 2. Ascorbic acid 5 %. (50 mg/ml) 3. Butomidor (10 mg/ml)
Third experimental (E3) 1. Schiff-Sherrington syndrome is caused by an injury to the vertebrae of the thoracic spine 2. Root syndrome	5	1. Solu-Medrol (1000 mg/ml) 2. Alpha-tocopherol acetate 30% (300 mg/ml) 3. Mannitol 15 % (150 mg/ml)

It is worth noting that the composition of the control and three experimental groups is as follows:

Control group (n = 5)**:** German Shepherd, 4 years old, male; German Shepherd, 5 years old, male; German Shepherd, 4 years old, female; German Shepherd, 8 years old, male; German Shepherd, 9 years old, male.

Experimental group (E1), consisting of five animals (n = 5): Central Asian Shepherd, 3 years old, female; Central Asian Shepherd, 4 years old, female; German Shepherd, 5 years old, male; French Bulldog, 7 years old, female; Pug, 5 years old, male.

Experimental group (E2), consisting of five animals (n = 5): French bulldog, 4 years old, male; Rottweiler, 7 years old, male; German shepherd, 7 years old, male; short-haired dachshund, 4 years old, male; German shepherd, 6 years old, male.

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Experimental group (E3), consisting of five animals (n = 5): Shorthair Dachshund, 5 years old, male; German Shepherd, 6 years old, male; Shorthair Dachshund, 6 years old, male; German Shepherd, 5 years old, male; French Bulldog, 8 years old, female.

After determining the degree of spinal damage, all experimental animals were immobilised as indicated, especially in cases of vertebral fractures or instability. As a result of a compression fracture of the vertebral bodies with subsequent displacement of the fragments in different directions, complications in healing of the fracture site and formation of bone callus are possible over time. To immobilise the dogs, we used a board with holes from which wide bands extend to fix the patient's body in the area of the shoulder blades and the large acetabulum. Thus, it ensures the stability of the spine relative to the site of its injury.

Clinical testing was conducted on 15 animals aged 3 to 9 years (Table 3.20). The pharmacological effects of the drugs are described below.

"ALPHA-TOCOPHEROL ACETATE contains the active ingredient vitamin E acetate, which in terms of 100% dry matter corresponds to 300 mg in 1 ml of the drug for oral administration. Vitamin E is an antioxidant agent that slows down lipid peroxidation, which is activated in many diseases. In case of vitamin E deficiency in dogs, degenerative changes in nervous tissue, muscles and hepatocytes are observed. As part of a complex treatment, it is used for spinal cord injuries, as well as for diseases of the nervous system requiring antioxidant therapy. It is a fat-soluble vitamin.

Alpha-tocopherol acetate was administered orally at 100 mg per day (8 drops) once daily for 30 consecutive days. A repeat course of drug administration was performed after 3 months.

30 % solution for injection was administered 1 ml intramuscularly to small and medium-sized dogs 2-3 times a day, depending on the neurological deficit, and 2 ml intramuscularly to large dogs for a total course of 2-3 days. The concentration of alpha-tocopherol acetate in the body was maintained by oral administration of the solution after intramuscular injections at the time of receiving SCI (spinal cord injury).

"Ascorbic Acid 5% in 2 ml solution contains 100 mg of the active ingredient

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ascorbic acid. The vitamin takes an active part in redox reactions; it exhibits antioxidant properties, thus ensuring stability of cell membranes. It is involved in glucose metabolism and synthesis of steroid hormones. It is not synthesised in animals and belongs to water-soluble vitamins.

Ascorbic Acid was administered in 2 ml (100 mg) intramuscularly or intravenously to small and medium-sized dogs once daily for 7 consecutive days. In large breed dogs, the drug was administered at 4 ml (200 mg) intramuscularly or intravenously once daily for 7 consecutive days.

"Vitaxon belongs to the pharmacotherapeutic group of vitamin B1 in combination with vitamin B6 and vitamin B12. 2 ml of the solution contains thiamine hydrochloride in terms of 100 % dry matter 100 mg, pyridoxine hydrochloride in terms of 100 % dry matter 100 mg and cyanocobalamin in terms of 100 % dry matter 1 mg.

Neurotropic B vitamins have a beneficial effect in inflammatory and degenerative nerve diseases. They are used to eliminate deficiency conditions, and in large doses they have analgesic properties and normalise the functioning of the nervous system. In the body, vitamin B1 is phosphorylated to form biologically active thiamidine phosphate (cocarboxylase) and thiamine triphosphate (TTP). The latter, as a coenzyme, is involved in important functions of carbohydrate metabolism, which are crucial for metabolic processes of nervous tissue, in particular, affecting the conduction of nerve impulses in synapses. The main indications for use are the treatment of radicular syndrome and neuritis of various origins. It belongs to water-soluble vitamins.

Vitaxon was administered deeply intramuscularly in the first 3 days to small and medium-sized dogs at a dose of 1 and 2 ml, respectively, and 3 ml intramuscularly in the first 3 days to large dogs. To continue treatment and maintain the therapeutic effect, the drug was administered 2-3 times a week for a total course of 14-21 days.

"Prednisolone-Darnitsa (PREDNISOLONE-DARNITSA) belongs to the pharmacotherapeutic group of glucocorticosteroids for systemic use and contains prednisolone sodium phosphate (prednisolone) in 1 ml of solution in terms of prednisone - 30 mg. It has anti-inflammatory, antitoxic, antishock and immunosuppressive effects. In high doses, it increases the excitability of brain tissue

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and helps to reduce seizures. The main indications for use are traumatic, anaphylactic and surgical shock, severe allergic and anaphylactic reactions.

Prednisolone was administered for intervertebral disc injuries of the thoracolumbar spine subcutaneously at a dose of 1 mg/kg body weight twice daily for 3 days. For the next 3 to 5 days, the dose was reduced to 0.5 mg/kg per day (113,132, 133).

"MANNITUM-NOVOFARM is an osmolar diuretic solution containing 150 mg of mannitol per 1 ml of solution. The drug has a pronounced diuretic effect, promotes rapid removal of fluid from the vascular bed, and increases renal blood flow. Thus, as a result of administration, the volume of circulating blood increases, which has a diuretic effect and reduces intracranial pressure. The main indications for use are cerebral edema, cerebral hypertension, intensive care of convulsive states.

Mannitol 15% was administered for acute spinal cord and spinal cord injuries at a dose of 500 mg/kg for 30-60 minutes. In some cases, it was administered again after 6 hours intravenously. In delayed clinical conditions with axial skeletal injuries, the drug was administered at a dose of 250 mg/kg over 30 minutes. It should be noted that mannitol solution 15% was not administered in coma.

"SOLU-MEDROL in 1 vial contains prednisone sodium succinate (methylprednisolone), equivalent to 1000 mg of methylprednisolone, and is a corticosteroid for systemic use. The highly concentrated solution is used to treat conditions that require rapid action of the hormone. It has a strong and long-lasting anti-inflammatory, anti-allergic and immunosuppressive effect. The drug has an effect on the cardiovascular system, skeletal muscles and central nervous system. Indications for the administration of Sol-Medrol are acute spinal cord injury, cerebral edema after a spinal cord injury. The effect of the drug is realised through the steroid receptor complex, which is transported to the cell nucleus and alters gene transcription for proteins, thus causing the destruction of cytokines, which play an important role in immune and inflammatory reactions. Methylprednisolone sodium succinate has virtually no mineralocorticoid activity and is a synthetic drug.

Solu-Medrol was administered at a dose of 30 mg/kg intravenously, followed by

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a repeat dose of 15 mg/kg intravenously in 2 hours and 10 mg/kg 4 times daily for 3 consecutive days after 6 hours, with a gradual reduction in the dose.

Manufacturer: Pfizer, Belgium.

"Armadin belongs to cardiological drugs and drugs affecting the central nervous system and contains 100 mg of ethylmethylhydroxypyridine succinate in 2 ml. The drug has cytoprotective, angioprotective, nootropic, antianginal, neuroprotective, anti-ischemic, antihypoxic effects, inhibits free radical lipid oxidation processes and increases the activity of enzymes of the body's antioxidant system, reduces the manifestations of oxidative stress in the body. It improves neurotransmitter transport and synaptic transmission.

Armadin was administered by slow drip infusion in sodium chloride saline for 30-90 minutes (2-3 ml/min) at a dose of 100 mg in small dogs, 200-300 mg intravenously in medium and large dogs, depending on the severity of the neurological deficit.

"Butomidor is an analgesic of the synthetic opioid group with agonist-antagonist effect. The drug has analgesic and sedative effects with a mild to moderate sedative effect. 1 ml of the product contains the active ingredient butorphanol in the form of hydrogen tartrate in the amount of 10 mg. The main indications for administration are pain relief before and after surgery and as an analgesic.

The drug Butomidor was administered intramuscularly at a dose of 0.25 mg/kg body weight for analgesic effect and 0.4 mg/kg body weight for sedation in the form of intravenous injection. The sedative effect was observed in the body for up to 16-18 hours. The frequency of the drug administration depended on the indications and lasted from 3 to 5 injections, once a day.

Manufacturer: Richter Pharma AG, Austria.

"DEPO-MEDROL is a corticosteroid for systemic use and contains 40 mg of methylprednisolone acetate in 1 ml of sterile suspension. It has a strong and long-lasting immunosuppressive, anti-inflammatory and anti-allergic effect. The long-lasting effect of Depo-Medrol is due to the slow release of the active substance. The drug has an effect on the cardiovascular system, skeletal muscles and central nervous

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system. It should be noted that the drug is not indicated for the treatment of acute conditions that directly threaten the life of the animal.

The main indications for use were delayed clinical conditions in dogs, where the drug was considered as a means of symptomatic and adjunctive therapy for tissue edema, allergic conditions and spinal cord and spinal cord contusions.

Depo-Medrol was administered at a dose of 1 mg/kg body weight intramuscularly once a week in the amount of 2 to 3 injections. The frequency of the drug administration depended on the degree of spinal and nerve damage and neurological deficit during the neurological examination. The medicine is not intended for intravenous administration.

Manufacturer: Pfizer, Belgium.

The effectiveness of drug treatment was assessed based on the results of the neurological status of the experimental animals, as well as biochemical and morphological blood tests. During long-term treatment with glucocorticosteroid drugs, 15 % of animals develop gastrointestinal bleeding, of which 2 % may die. H2-receptor antagonists were used to prevent the development of oesophageal reflux, gastric ulcers, uremic and erosive gastritis caused by pharmacological agents. Quamatel (active ingredient famotidine) competitively inhibits histamine, reducing both baseline gastric acid secretion and stimulation by feed, pepsinogastrin and histamine. It has no effect on the evacuation time of gastric contents, bile secretion and pressure in the lower oesophagus. As a result, the secretion of pepsin decreases. The dose of the drug was 0.5 mg/kg by intravenous injection in isotonic sodium chloride solution 0.9% with an interval of 12 hours, 2 times a day for up to 5 consecutive days [132]. The main recommendation for animal owners was to carefully administer the drug to ensure clinical effect. Otherwise, the symptoms of gastroesophageal reflux or erosive gastritis could progress to the stage of relapse.

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2.4. Testing of methods of surgical treatment of animals with neurological syndromes in acute spinal injuries

After a clinical examination of the animals, followed by a determination of the neurological deficit and the degree of spinal damage, immobilisation was often used to transport the dogs, followed by fixing the torso to a board to prevent excessive movement.

For the neurological examination of dogs, the Neurological Examination Form (2010) protocol was used, which takes into account the criteria with possible pathological changes in the nervous system that determine the localisation of the process; the Griffiths (1982) scale of neurological disorders to determine the sensitivity of the limbs (Table 3.4); to assess the degree of neurological disorders, the scale by Scott H. W, McKee W. M. (1999), which was used to determine the presence of pain syndrome, the absence or presence of pain sensitivity, and the establishment of signs of paresis and paralysis of the limbs (Table 3.6); the neurological scale Thoracolumbar injury classification and severity score (TLICS) to assess the degree of spinal injury and determine the methods of conservative or surgical treatment (Table 3.21) and the neurosurgical scale for determining the gradation of sensory and motor function according to the American Spinal Injury Association (ASIA).

Experimental group (E1), consisting of five animals (n = 5): Central Asian Shepherd, 3 years old, female; Central Asian Shepherd, 4 years old, female; German Shepherd, 5 years old, male; French Bulldog, 7 years old, female; Pug, 5 years old, male.

The complex of drugs used: Depo-Medrol + Vitaxon + Armadin.

In 5 experimental dogs, the main pathological complex was diagnosed in the form of lumbosacral syndrome at the level of the seventh lumbar (L7) - first sacral (S1) vertebrae, which in turn causes the development of the "cauda equina" syndrome and the caudal spinal cord injury syndrome.

The main method of surgical treatment of lumbosacral syndrome and cauda equina syndrome is lumbosacral hemilaminectomy (described below). In three dogs,

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the technique was performed in combination with a foramenectomy, which allowed access to the articular surfaces of the vertebrae and intervention at the level of the intervertebral foramen in case of spinal root compression. It is also possible to resect osteophytes that cause nerve compression (Fig. 3.16). In two animals, a combined surgical technique was performed in the form of hemilaminectomy with facetectomy, characterised by the removal of articular surfaces [113]. The manipulation is performed by compressing the nerve root to identify the appropriate intervertebral foramen, and then removing the articular processes with a drill or Luer forceps. In cases of vertebral body fractures in the lumbar spine, additional stabilisation is performed (Figs. 3.17-3.18).

After the surgical intervention, the dogs were restricted in movement for 4-5 weeks, depending on the manifestation of neurological dysfunctions. All animals received a course of glucocorticosteroid treatment: Depo-Medrol (40 mg/kg). To prevent complications from the gastrointestinal tract, Kvamatel (famotidine) was administered. The dose of the drug was 0.5 mg/kg by intravenous injection in isotonic sodium chloride solution 0.9% with an interval of 12 hours, twice a day for up to 5 consecutive days [132].

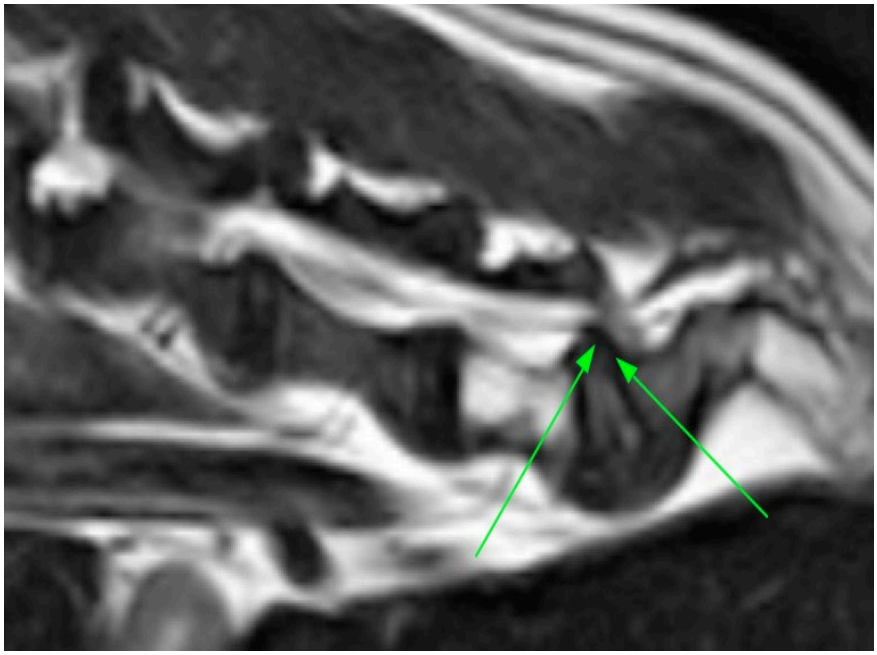

Fig. 3.16 - MRI scan with Cauda equina syndrome at the level of the seventh lumbar vertebra (L7) in a 7-year-old French bulldog, nicknamed Teddy (E1). Sagittal plane

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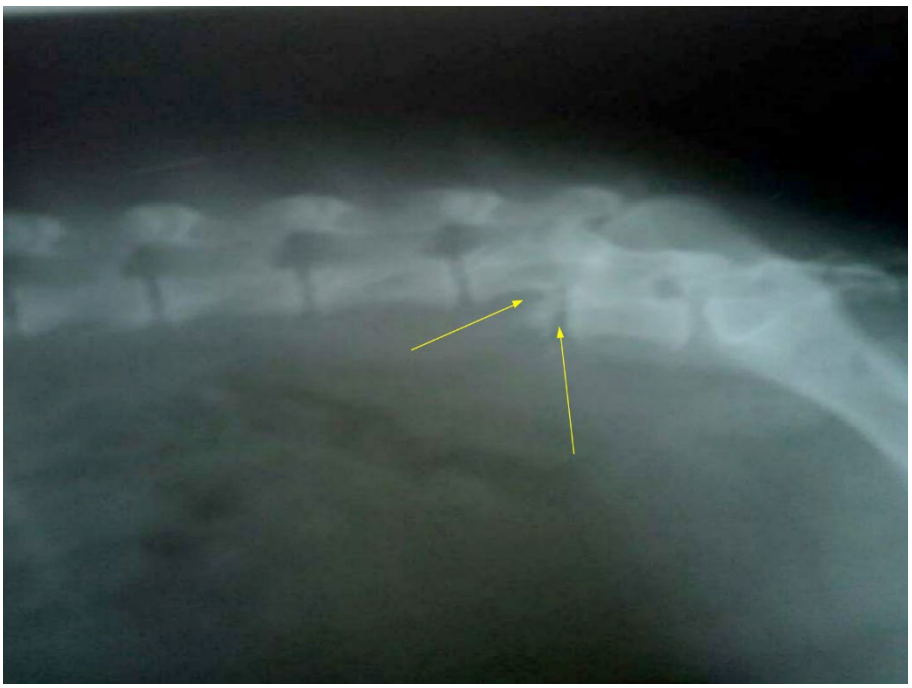

Fig. 3.17 - X-ray of a German Shepherd, John, aged 5 years (E1) with a ligamentous complex disruption and compression fracture at the level of the fifth to sixth lumbar vertebrae (L5 - L6). Lateral projection

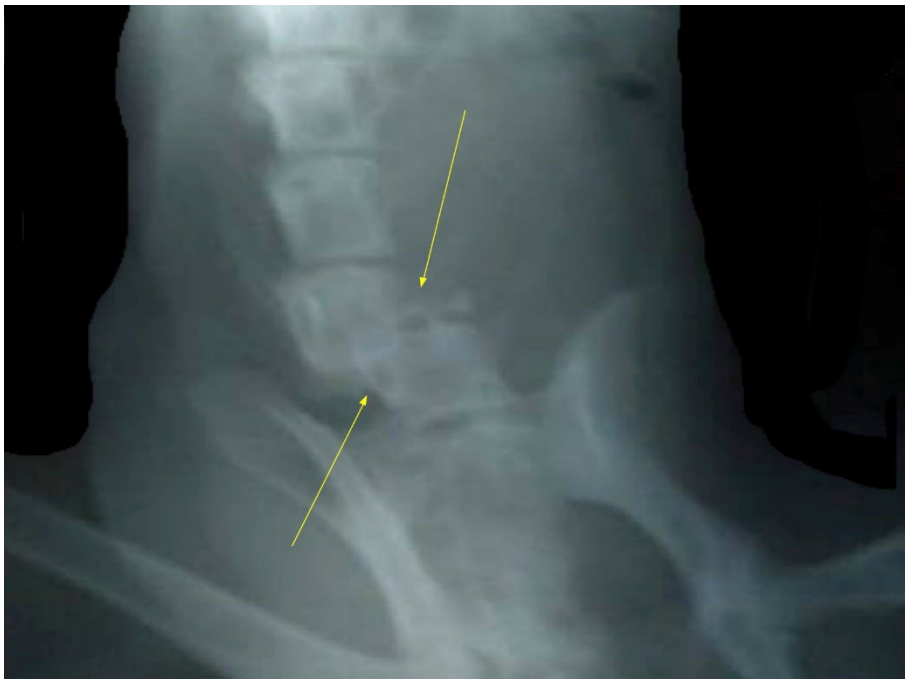

Figure 3.18 - Radiograph of John, a German Shepherd Dog, 5 years old (E1) with a rotational and compression fracture at the level of the fifth to sixth lumbar vertebrae (L5 - L6). Dorsal projection

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Experimental group (E2), consisting of five animals (n = 5): French bulldog, 4 years old, male; Rottweiler, 7 years old, male; German shepherd, 7 years old, male; short-haired dachshund, 4 years old, male; German shepherd, 6 years old, male.

The complex of drugs used: Prednisolone + Ascorbic acid 5% + Butomidor.

In dogs diagnosed with Hansen type 1 intervertebral disc disease, surgery was performed as described. Hemilaminectomy or mini-hemilaminectomy, depending on the breed and size of the dog, was performed using a lateral or dorso-lateral approach to the spine with the dog in a lateral or semi-lateral position [113]. In the dorso-lateral approach, the longest muscle was separated from the multifidus muscles of the lumbar spine in the craniomedial direction, where the tendon of the longest muscle joins the accessory processes of the vertebrae. Sections of the intervertebral disc and vertebral arch were separated from the muscles and the tendon of the longest muscle was dissected. An additional vertebral process was removed with the help of a cutter, followed by resection of the vertebral arches to create a hole 1/2-2/3 of the length of each vertebra. To stop bleeding from the venous sinuses or vertebral artery, a bipolar coagulator with the following characteristics was used: bipolar coagulation - 80 W/100 Ω; bipolar cutting - 80 W/200 Ω. Minor bleeding was eliminated by applying a haemostatic sponge with amben. The cortical and spongy parts of the vertebrae were cut out evenly along its entire length with a high-speed cutter, preventing its "falling through" at a certain point. A physiological solution of sodium chloride was used to cool the bone. After the spongy bone was removed, the inner cortical layer was thinned to a thin, mobile state. After its puncture, access to the spinal canal was opened. Kerrison's nippers were used to remove the remnants of the vertebral cortical layer. A portable aspirator was used to remove the remains of intervertebral discs. After removing the latter, the spinal cord was returned to its anatomical position. In case of its displacement, it was necessary to determine the places of additional compression on the opposite side of the spine and carefully review the MRI images to determine the exact location of the intervertebral disc extrusion or formed exostoses (Figs. 3.19-3.20).

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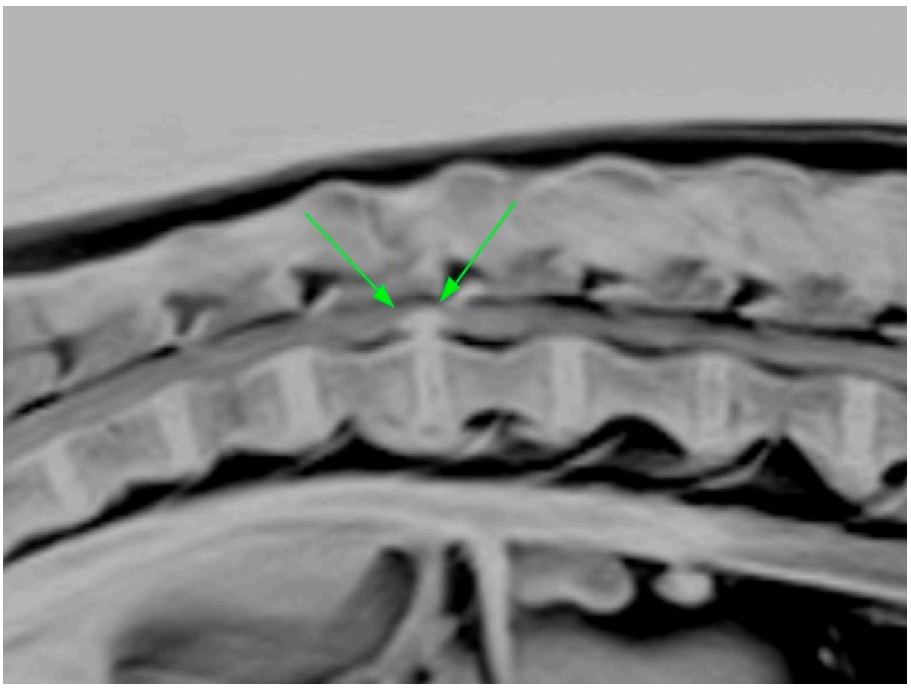

Fig. 3.19 - MR imaging with intervertebral disc extrusion in a 4-year-old dachshund named Phil (E2). The image was taken in the negative. Sagittal plane

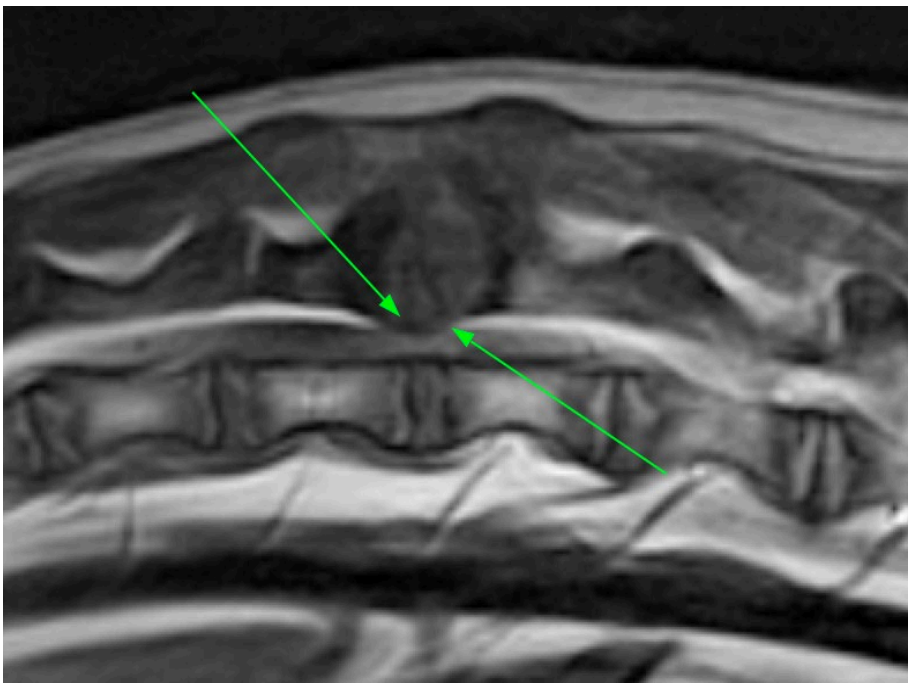

Fig. 3.20 - MR imaging with bone post-traumatic exostosis in a 7-year-old German shepherd dog, Claude (E2), with manifestations of radicular syndrome. The joints are articulated. Sagittal plane

After the surgery, the dogs were restricted in movement for 5-7 weeks, depending on the clinical manifestation of the neurological deficit. All animals

received a course of glucocorticosteroid treatment: Prednisolone (30 mg/kg). To prevent complications from the gastrointestinal tract, Quamatel (famotidine) was administered. The dose of the drug was 0.5 mg/kg by intravenous injection in 0.9% isotonic sodium chloride solution with an interval of 12 hours, twice a day for up to 5 consecutive days [132].

Experimental group (E3), consisting of five animals (n = 5): Shorthair Dachshund, 5 years old, male; German Shepherd, 6 years old, male; Shorthair Dachshund, 6 years old, male; German Shepherd, 5 years old, male; French Bulldog, 8 years old, female.

The complex of drugs used: Solu-Medrol + Alpha-tocopherol acetate + Mannitol 15%.

The Thoracolumbar injury classification and severity score (TLICS) scale was used to reliably assess the degree of spinal injury and determine methods of conservative or surgical treatment (Table 3.21).

The following data were obtained using the neurological scale (TLICS):

Short-haired dachshund, 5 b.: 7 b.; German Shepherd, 6 b.: 7 b.; short-haired dachshund, 6 b.: 8 b.; German Shepherd, 5 years old: 9 b.; French Bulldog, 8 b.: 7 б. Summing up the indicators, we obtain results that indicate the expediency of surgical intervention after conservative treatment.

Early spinal cord decompression is a prerequisite for the successful treatment of patients with spinal cord injuries [113]. Numerous experimental studies have proven the effectiveness of high doses of methylprednisolone sodium succinate, as it has the ability to reduce spinal cord edema, restore electrolyte balance, and improve post-traumatic cerebral blood flow [78]. It is much more effective than prednisolone and dexamethasone because it is an inhibitor of lipid peroxidation. Dogs underwent surgical intervention with vertebral fixation with a plate and the use of polymethacrylate bone cement. Intraoperative view of the spinal cord: acute spinal cord edema and partial disruption of its integrity.

Table 3.21 - TLICS scale classification

Morphology: the mechanism of injury		
Compression (simple compression)	-	1 point
Explosive compression	-	2 points
Progressive/Oberthoracic	-	3 points
Distraction	-	4 points
Integrity of the communication complex		
Whole	-	0 points
Suspect/undetermined	-	2 points
Damaged	-	3 points
Neurological status		
Norma	-	0 points
Damage to the roots	-	2 points
Spinal cord cone - normal	-	2 points
Spinal cord cone: damaged	-	3 points
Ponytail	-	3 points
Methods of treatment		
3 or less points:	conservative treatment	
4 points:	choice of conservative or surgical treatment	
5 or more points:	surgical treatment	

During the entire observation period, all animals were provided with high-quality food and watering. To varying degrees, some dogs consumed the food on their own, while others were fed with the help of a veterinarian or with a pre-installed nasogastric tube. The main canned food to restore the body's defences for dogs in the second and third experimental groups: Royal Canine Recovery. Other animals were fed Purina Vet Diet Convalescence.

After the surgery, the dogs were restricted in movement for 5-8 weeks, depending on the clinical manifestation of the neurological deficit. All animals received a course of glucocorticosteroid medication: Solu-Medrol. To prevent

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complications from the gastrointestinal tract, Kvamatel (famotidine) was administered. The dose of the drug was 0.5 mg/kg by intravenous injection in isotonic sodium chloride solution 0.9% with an interval of 12 hours, 2 times a day for up to 5 consecutive days [132].

2.5. Haematological parameters in dogs during treatment with different methods

The effectiveness of the tested treatment methods was assessed by regular clinical examination of experimental animals and monitoring of their neurological status.

For an objective assessment of the clinical condition and efficacy of therapeutic regimens in animals of the experimental groups (E1, E2, E3) and dogs of the control group (no treatment), haematological studies were performed. Blood samples were taken from the first day of the experiment and the application of conservative treatment on the 1st, 5th, 14th, 30th and 55th day, respectively, from the moment of the initial administration of animals.

The results of the study of the number of leukocytes, erythrocytes, hematocrit and haemoglobin content in dogs of the control and experimental groups during the treatment period are given in Table 3.22.

The number of **erythrocytes** in the blood of animals of *the control* group undergoing self-healing from day 1 to day 14 of the study corresponded to the parameters of the lower limit of normal. Already on the 30th day of the experiment, it acquired the average values of the range of physiological fluctuations, and on the 55th day it again underwent changes that were numerically close to the lower limit of normal.

In animals *of the first* experimental group treated with the complex of drugs depo-medrol + vitaxone + armadine, the number of red blood cells in the blood changed significantly only on the 30th day of the experiment and was characterised by a decrease in its value by 12.1 % compared to the control. At the same time, in dogs *of the second* experimental group (treatment with a complex of drugs prednisolone +

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ascorbic acid, 5% + butomidor), this indicator was higher than the control values on the 1st, 5th and 14th day of the study (approaching the maximum parameters of the norm), respectively, by 42.9%, 53.4% and 26.6%, which may be the result of red blood cell mobilisation from the blood depot and reflects the development of positive compensatory changes in the body under the appropriate therapeutic regimen.

Table 3.22 - Formal blood elements, haemoglobin and haematocrit value in experimental dogs (M ± m, n = 5 - 15)

Day of experiment	Group.	Red blood cells, $10/l^{12}$	Haemoglobin, g/l	Hematocrit, %	White blood cells, $10/l^{9}$
		Reference values for norms [133, 134].			
		5.0-8.5	120-190	37-55	6.0-9.4
1st	C	5.6±0.2	123.4±0.7	40±1	7.7±0.2
	E1	5.4±0.2	158.4±0.3**	39±1	10.8±0.2*
	E2	8.0±0.2*	190.8±0.2*	35±1*	13.6±0.2*
	E3	3.7±0.2*	82.6±0.3*	29±1*	17.1±0.5*
5th	C	5.5±0.2	125.4±0.6	41±0.1	7.9±0.2
	E1	5.5±0.3	164.8±0.2*	38±0.6*	11.4±0.2*
	E2	8.5±0.2***	190.8±0.1*	32±0.7*	13.9±0.3*
	E3	3.8±0.2**	84.4±0.3***	28±0.7*	17.3±0.4*
14th	C	6.3±0.3	137.8±0.3	40±0.8	7.6±0.2
	E1	6.0±0.2	163.4±0.4*	35±0.6*	11.6±0.1*
	E2	7.9±0.1*	190.6±0.1*	30±0.7*	14.5±0.2*
	E3	4.2±0.2*	74.2±0.4*	28±0.6*	16.5±0.3*
30th	C	6.8±0.3	145.4±0.6	43±0.9	7.2±0.2
	E1	6.0±0.1*	167.0±0.4*	32±0.3*	11.2±0.3*
	E2	6.8±0.3	188.0±0.2**	27±0.4*	14.7±0.3*
	E3	4.3±0.3*	71.0±0.2*	26±0.7*	16.3±0.4*
55th	C	6.2±0.3	145.6±0.4	42±1.3	7.2±0.2
	E1	6.3±0.2	160.2±0.1*	39±0.7*	9.2±0.**2****
	E2	6.0±0.1	170.8±0.1**	41±0.1	11.7±0.3*
	E3	5.4±0.4	114.6±0.2**	37±0.6**	11.7±1.1**

Note: * p≤0.001, ** p≤0.01, *** p≤0.05 compared to the values of the control group of dogs; C - control, E1, E2, E3 - experimental groups

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In turn, in dogs *of the third* experimental group, there was a decrease in the number of erythrocytes from day 1 to day 30 of the experiment inclusive and corresponded to values lower than the control, respectively by 33.9 %, 31.5, 33.0 and 36.7 %. Based on the experimentally established patterns, it should be noted that this indicator undergoes the most pronounced changes in the last group of animals, which may be due to inhibition of erythrocytopoiesis in the red bone marrow of severely ill dogs. At the same time, the use of a complex of drugs (sol-medrol + alpha-tocopherol acetate + mannitol 15 %) contributed to the restoration of this indicator on the 55th day of the experiment.

The **haemoglobin** content in the blood of *the control* group of animals from day 1 to day 14 of the study corresponded to the lower limit of normal. On the 30th and 55th day of treatment, the obtained values reached the average values of the range of physiological fluctuations (see Table 3.22). The lowest haemoglobin content was found in the blood of these animals on days 1 and 5, indicating the development of iron deficiency anaemia as a result of prolonged anorexia with severe pain due to spinal cord compression.

In the animals of *the first* experimental group treated with the complex (depot-medrol + vitaxone + armadin), the haemoglobin content increased by 28.4%, 31.4%, 18.6%, 14.9% and 10.0%, respectively, compared to the control from day 1 to day 55 of treatment, indicating the development of hyperchromemia, which reduces the negative effect in the event of posttraumatic anaemia.

The most pronounced changes in the blood haemoglobin content were observed in animals *of the second* experimental group, which consisted of its increase on the 1st, 5th and 14th day, respectively, by 54.6 %, 52.1 and 38.3 % compared to the control, and a slight decrease in the future. However, its level remained high both on the 30th and 55th day of the study (by 29.3% and 17.3%, respectively). This is an important compensatory reaction of the body aimed at maintaining the gas balance in the body of animals with severe spinal pathology.

In dogs *of the third* experimental group, on the 1st day of the study, the level of haemoglobin in the blood was 54.6 % of the control group, indicating the development

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of hypochromic anaemia. Low values of this indicator were also noted in the dynamics of pathology development on the 5th, 14th and 30th day of treatment, respectively, 32.7%, 46.2% and 51.2% less than the control, which confirms the progression of anaemia, which may be a consequence of acute blood loss with partial or complete spinal cord rupture and impaired integrity of large-calibre blood vessels, endogenous intoxication with tissue decay products in spinal cord injury. At the same time, on the 55th day of complex treatment (solu-medrol + alpha-tocopherol acetate + mannitol 15 %), the hemoglobin content was characterized by an increase of 21.3 % and reached the level of the lower limit of the norm, indicating a partial restoration of the function of hematopoietic organs with subsequent normalisation of the clinical condition of animals during the rehabilitation period (see Table 3.22).

The value of **hematocrit** in the animals of *the control* group during the entire study period from day 1 to day 55 corresponded to the values of the lower limit of the norm. The determined parameters in dogs were in the range of 40 - 43 %, which corresponds to the development of a chronic pathological process in them as a result of trauma.

In animals of *the first* experimental group (complex treatment with depot medrol + vitaxone + armadine) (see Table 3.22), the haematocrit value was characterised by lower values on days 5-55 of treatment, respectively by 7 %, 12.5, 25.6 and 7 % compared to the control. This confirms the development of anaemia in traumatised dogs, and may also be a consequence of prolonged starvation of experimental animals as a result of severe pain syndrome with lumbosacral instability of the lumbar spine vertebrae. On the 55th day, a slight increase in its value was noted, indicating a gradual recovery of the animal's condition after surgery and vertebral stabilisation.

In dogs *of the second* experimental group, a significant decrease of 22.0 % in the haematocrit value on the 5th day of treatment was found, which may be the result of hydremia due to haemodilution by using infusion saline electrolyte solutions. Accordingly, on the 14th and 30th day of treatment, the haematocrit value was determined at 25.0 and 37.2%, respectively, indicating a critical state of health of the experimental dogs. As a result of the use of the complex treatment regimen

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(prednisolone + ascorbic acid 5% + butomidor) on the 55th day of the experiment, the recovery of the hematocrit value was established, indicating a temporary improvement in the clinical condition of dogs.

In the animals *of the third* experimental group, a decrease in haematocrit value was observed on the 1st, 5th and 14th day of complex treatment, respectively by 27.5 %, 31.7 and 30.0 % compared to the control. This is due to the fact that high doses of a glucocorticosteroid drug were administered intravenously to experimental animals, which has an inhibitory effect on the body's immune response in the pathological process with spinal cord injury. The most pronounced decrease in the value of the indicator (by 39.5 %) was observed on the 30th day of treatment of dogs of this group and its probable increase on the 55th day (only 12 % less than the control level). The complex of neurological syndromes is quite dangerous for the life of patients and threatens with death within 3-5 days after spinal cord injury.

The number of **leukocytes** in the blood of dogs in *the control* group remained within the reference norm throughout the treatment period (see Table 3.22).

In the animals *of the first* experimental group treated with the complex (depo-medrol + vitaxone + armadin), a significant increase in the number of leukocytes by 40.3 % was found in the blood on the 1st day of treatment compared to the control (see Table 3.22). It should be noted that on the 5th day of the study, a significant increase in the number of leukocytes (by 44.3%) was detected compared to the control, which may be due to the manifestation of pain and inflammation. This trend persisted on the 14th and 30th day of the study with an increase in this indicator by 53.0 and 55.6 %. Leukocytosis may also be a consequence of intoxication and allergic reactions in the diseased animals. On the 55th day of treatment, the number of leukocytes in the blood reached the upper limit of normal, indicating a gradual recovery of experimental dogs of the first group during 14-20 days of the rehabilitation period. From the 1st to the 55th day of the study, the number of leukocytes in the blood remained significantly higher than that of the control group.

In animals *of the second* experimental group treated with a complex of drugs (prednisolone + ascorbic acid 5% + butomidor), a pronounced leukocytosis was

observed at all stages of conservative treatment.

On the 1st and 5th day, a significant increase in the number of leukocytes by 76.6 and 75.9 % was found compared to the control. Particularly pronounced leukocytosis in experimental animals of this group was noted on the 14th and 30th day of treatment, which was characterised by an increase in the number of leukocytes by 90.8 and 104.1 % compared with the corresponding control. During this period, glucocorticosteroid and antioxidant agents were administered to prevent the progression of ascending syndrome. It is worth noting that the ascending myelomalacia syndrome is characterised by spinal cord lysis and soft tissue decay products accumulate in the spinal canal, which enter the brain through the cerebrospinal fluid circulation. On the 55th day of treatment, the number of leukocytes remained 62.5% higher than the upper limit of normal compared to the control. As a result, the prognosis was determined as cautious to unfavourable (see Table 3.22).

In dogs *of the third* experimental group, a pronounced leukocytosis was found in the period from the 1st to the 30th day of treatment, characterised by the highest intensity of increase in the number of leukocytes in the blood, respectively by 122.0%, 119.0, 117.1 and 126.3% compared to the control group. The highest rates were recorded on the 1st day after spinal cord injury with the subsequent development of spinal shock and on the 30th day, which is explained by the administration of high doses of glucocorticosteroid drugs. In turn, the development of a concomitant neurological syndrome inhibits the recovery of their number during treatment, as periodic clonic seizures contributed to the exacerbation of the underlying disease. The use of the proposed complex of therapeutic agents contributed to the restoration of nerve impulses and reduced the formation of lipid peroxide compounds at the site of spinal cord injury. On the 55th day, the number of leukocytes was still high relative to the upper limit of normal. At the same time, the prognosis for the restoration of motor function in injured dogs remained from cautious to unfavourable.

From day 1 to day 55 of the study, the number of leukocytes was significantly higher than in the control group (see Table 3.22).

Changes in the blood leucogram of dogs of the control and experimental groups

during the treatment period are shown in Table 3.23.

Table 3.23 - Blood leukogram in experimental dogs (M ± m, n = 5 - 15)

Group.	Leukogram, %.							
	Basophils	Eosin-ophils	Neutrophils				Lymphocyt-es	Monocytes
			Myelocytes	Metamyelocyte	Band	Segmented		
	Reference values for norms [133, 134].							
	-	0-7	-	0-1	0-4	60-70	12-30	2-10
1st day								
C	-	1.8±0.4	-	-	2.0±0.3	68.0±1.3	21.2±1.1	7.0±0.3
E1	-	2.8±0.4	-	-	3.0±0.3*	64.8±2.4	20.6±1.6	8.8±0.7*
E2	-	3.4±0.3*	-	-	7.4±0.3*	61.6±1.3**	19.8±1.4	7.8±0.4
E3	-	3.8±0.4*	-	-	9.2±0.4*	52.4±1.8*	24.8±1.2*	9.8±0.4**
Day 5								
C	-	2.4±0.3	-	-	2.4±0.3	68.4±1.3	20.0±1.0	6.8±0.4
E1	-	3.6±0.6*	-	-	3.6±0.6*	62.2±2.0*	21.0±1.0	9.8±0.4*
E2	-	3.6±0.6*	-	-	6.8±0.4*	61.0±1.0**	20.4±1.3	8.2±0.4*
E3	-	3.2±0.4	-	-	7.2±0.7*	53.2±9.6*	24.4±0.6**	12.0±0.3*
14th day								
C	-	3.8±0.4	-	-	2.6±0.3	67.0±2.0	19.6±1.6	6.2±0.4
E1	-	5.0±0.3*	-	-	4.0±0.3**	67.6±1.0	12.8±0.4*	9.4±0.7**
E2	-	4.8±0.4	-	-	5.8±0.4*	66.0±1.3	16.4±0.6*	6.8±0.4
E3	-	4.6±0.7	-	-	7.8±0.4***	69.6±0.8	11.2±0.4**	6.2±0.4
30th day								
C	-	2.8±0.4	-	-	2.2±0.4	67.0±1.5	18.4±1.7	7.6±0.6
E1	-	4.0±0.3*	-	-	4.2±0.4*	68.8±2.5	12.4±0.6*	8.6±0.8
E2	-	5.4±0.6**	-	-	4.8±0.4**	69.4±1.2	14.4±0.6*	5.6±0.3*
E3	-	5.8±0.4**	-	-	4.8±0.4**	74.6±0.6*	8.8±0.4*	5.4±0.3**
55th day								
C	-	2.6±0.6	-	-	2.8±0.4	66.4±1.0	20.8±0.7	7.4±0.6
E1	-	3.4±0.6	-	-	2.8±0.4	71.6±1.0*	15.2±0.7*	7.8±0.4
E2	-	3.2±0.7	-	-	3.4±0.6	67.2±1.9	18.2±0.9*	7.0±0.3
E3	-	3.4±0.5	-	-	4.4±0.4*	72.0±1.3*	12.6±0.6*	7.6±0.5

Note: * p≤0.001, ** p≤0.01, *** p≤0.05 compared to the values of the control group of dogs; C - control, E1, E2, E3 - experimental groups

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The relative number of **eosinophils** in the leukogram *of the control* group animals during the experiment was within the reference range.

In the dogs of *the first* experimental group, the relative number of eosinophils also corresponded to the range of the reference norm throughout the treatment period, but at each stage it was characterised by changes towards an increase. Thus, on the 5th, 14th, 30th and 55th day, the relative number of eosinophils in the blood of dogs increased by 1.5, 1.3, 1.4 and 1.3 times, respectively, compared to the control, which may be due to general intoxication of the body due to spinal cord injury.

In the animals of *the second* experimental group, on the 1st and 30th day of treatment, the relative number of eosinophils reached the highest values, in particular, 1.8 and 1.9 times compared to the control. This, in turn, indicates a side effect of the combined administration of a glucocorticosteroid drug in therapeutic doses with an analgesic to sick animals. On day 5, their relative number increased by 1.5 times compared to the control group of dogs.

In the animals of *the third* experimental group, on the 1st and 30th day of treatment, the relative number of eosinophils showed a similar trend and increased 2.1 times compared to the control. In other periods of the experiment, the indicators of the experimental groups remained unchanged.

No significant fluctuations in the relative number of neutrophils were observed in the blood of the control group dogs throughout the experiment.

The relative number of **rosette neutrophils** in the leukogram *of the control* group was within the average normal range. For comparison, in dogs of the first experimental group of animals, the average values increased by 1.5 times, in the second - by 2.4 times and in the third - by 2.8 times compared to the control group during the entire course of treatment (see Table 3.23).

In the animals *of the first* experimental group, which were comprehensively treated with depo-medrol + vitaxone + armadin, the relative number of rods of neutrophils in the leukogram was higher on the 1st, 5th and 14th day of treatment, respectively, by 1.5 times compared to the control (see Table 3.23). At the same time, on the 30th day of the experiment, this indicator showed maximum changes by 1.9

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times compared to the control. A moderate increase in the relative number of rods of neutrophils is explained by inflammatory processes in nerve root neuritis in the syndrome of caudal spinal cord injury. Already on the 55th day of treatment, the restoration of the relative number of rods neutrophils in the blood was noted.

In the animals *of the second* experimental group treated with a complex treatment (prednisolone + ascorbic acid 5% + butomidor), the relative number of these cells significantly exceeded their values in the control at all stages of treatment (in particular, on the 1st, 5th, 14th, 30th and 55th day, 3.7 times, 2.8 times, 2.2 times, 2.2 times and 1.2 times, respectively), which is due to the severe course of the pathological process with tissue necrosis and ascending myelomalacia, followed by the disintegration of the soft and hard membranes of the spinal cord, and severe intoxication. At the same time, the dynamics of changes in this indicator indicates positive changes in the clinical condition of animals and the effectiveness of the applied therapeutic regimen.

At the same time, in animals *of the third* experimental group treated with a complex of drugs (solu-medrol + alpha-tocopherol acetate + mannitol 15 %), the relative number of these cells significantly exceeded the control group, which confirms the presence of an inflammatory process of the spinal roots in the form of neuritis at the level of the thoracic and lumbar spine. During the course of treatment from the 1st to the 55th day, both severe and moderate neutrophilia was observed, which, according to the stage of the study, was 4.6 times, 3.0 times, 3.0 times, 2.2 times and 1.6 times higher than in the control group of animals (see Table 3.23). Based on this, dogs with spinal cord injury and partial spinal cord tear recover their interrelated functions rather slowly, which require specialist attention even 2 months after conservative and surgical treatment.

The relative number of **segmented neutrophils** in the blood leukogram of all experimental animals was within the reference norm (see Table 3.23).

At the same time, in dogs of *the first* experimental group, a decrease in the relative number of segmented neutrophils on the 5th day of treatment by 1.1 times and an increase by 1.1 times on the 55th day of the experimental study were found

compared to the control.

In the animals *of the second* experimental group, a decrease in the relative number of segmented leukocytes was observed only at the beginning of treatment, which differed by 1.1 times on both the 1st and 5th day of treatment compared to the control.

In the animals of *the third* experimental group, from day 1 to day 5 of treatment, the relative number of segmented neutrophils was characterised by a similar tendency to decrease, respectively by 1.3 times, and on days 30 and 55 it increased by 1.1 times, respectively, compared to the control group of animals, indicating the development of mild neutropenia at the beginning of treatment, which gradually turned into moderate neutrophilia.

Thus, spinal cord and spinal cord injuries in combination with spinal cord injuries are accompanied by neutrophilia with a regenerative shift of the nucleus to the left, which is especially pronounced in dogs of the second and third experimental groups.

The relative number of **lymphocytes** in the leukogram of experimental dogs showed significant changes from the 14th day of treatment (see Table 3.23).

In the *control* group of dogs, the content of lymphocytes was within the average normal range.

At the same time, in dogs of *the first* experimental group treated with complex treatment (depot medrol + vitaxone + armadine), the relative number of lymphocytes decreased on the 14th, 30th and 55th day of the experiment, which was 1.5 times, 1.5 times and 1.4 times, respectively, compared to the control. Lymphocytopenia in these animals is explained by both the use of a glucocorticosteroid drug and a decrease in the body's defences.

In the dogs *of the second* experimental group, the relative number of lymphocytes was characterised by a decrease, which was manifested from the 14th day of treatment. In particular, on the second week of treatment, it decreased by 1.2 times, on the 30th day by 1.3 times, and on the 55th day by 1.1 times compared to the corresponding control.

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In dogs *of the third* experimental group, lymphocytosis, as a protective reaction of the body, was observed on the 1st and 5th day of treatment. Subsequently, the relative changes in lymphocytes in the blood corresponded to the state of lymphocytopenia observed on the 14th, 30th and 55th day of the therapeutic complex (solu-medrol + alpha-tocopherol acetate + mannitol 15%), respectively, by 1.8 times, 2.1 and 1.7 times compared to the control group of animals. The latter indicates the development of an immunodeficiency state in traumatised dogs, which requires a long course of rehabilitation.

Summarising the results of the study, it can be concluded that not all stages of treatment of dogs with spinal cord and spinal cord injuries result in the development of lymphocytopenia. Thus, as a result of the studies, no decrease in the relative number of lymphocytes was recorded at the initial stage of treatment of the disease in various neurological syndromes in animals of all experimental groups. On the contrary, in the first and third experimental groups of animals, lymphocytopenia was observed from the 14th to the 55th day of treatment, indicating the need for medical support of the body's defences and long-term rehabilitation of such patients.

The relative number of **monocytes** and lymphocytes in the blood leucogram of all experimental dogs underwent certain changes (see Table 3.23).

In the animals of *the first* experimental group, the relative number of monocytes gradually increased by 1.3, 1.4 and 1.5 times, respectively, over two weeks compared to the control. Subsequently, their relative number returned to normal.

In dogs *of the second* experimental group, monocytosis (1.2-fold increase in the relative number of monocytes) was observed on the 5th day of treatment, and monocytopenia (1.4-fold decrease in the relative number of monocytes) was observed on the 30th day compared to the control. On the 55th day of the experiment, this indicator returned to normal.

In dogs *of the third* experimental group, on the 1st and 5th day of treatment, an increase in the relative number of monocytes was recorded, respectively, by 1.4 and 1.8 times compared to the control. On the 30th day of the experiment, a slight decrease in their number by 1.4 times compared to the control was noted in the blood of dogs of

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this group. Subsequently, this indicator was characterised by a gradual recovery, which may indicate the normalisation of the clinical condition and partial recovery of motor function in experimental animals.

The results of the analysis of peripheral blood leukograms of sick dogs indicate significant changes in the relative number of certain types of leukocytes during the recovery period after spinal injuries and in the case of treatment with approved drug complexes, which correlate in proportion to the severity of spinal injury.

The patterns of changes in the activity of enzymes in the blood plasma of experimental dogs are shown in Table 3.24.

According to the results of biochemical studies in *control* group animals during the study period, the activity of **AST** corresponded to the upper limit of the reference standard.

In the animals of *the first* experimental group treated with the complex (depot-medrol + vitaxone + armadine), the activity of AST increased on the 1st, 30th and 55th day of treatment, respectively, by 24.8%, 45.4% and 50.0% compared to the control, which is a manifestation of deep cellular damage due to mechanical spinal trauma and drug effects on internal organs, in particular the heart, liver and skeletal muscle.

In animals *of the second* experimental group, which were comprehensively treated with drugs (prednisolone + ascorbic acid 5% + butomidor), high values of plasma AST activity were observed on the 14th, 30th and 55th day of the study, respectively, by 58.2%, 59.5% and 46.0% compared to the control, characterised by a tendency to decrease only at the final stage of the therapeutic course.

In animals *of the third* experimental group treated with the complex (solu medrol + alpha-tocopherol acetate + mannitol 15%), the activity of AST also increased on the 14th, 30th and 55th day of the study, respectively, by 53.9%, 67.0% and 57.2% compared to the control, indicating positive changes and gradual restoration of the cellular organisation of damaged organs at the final stage of treatment of severely ill dogs.

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Table 3.24 - Plasma enzymes in experimental dogs (M ± m, n = 5 - 15)

Day of experiment	Group.	AST, Unit/l	ALT, Unit/l	Amylase, Unit/l	GGTP, Unit/l	Alkaline phosphatase, Unit/l
		Reference values for norms [133, 134].				
		5-40	9-42	25-640	1-6	50-90
1st	C	31.4±2.5	31.4±2.5	398.5±16.3	2.2±0.4	62.3±1.55
	E1	39.2±1.4*	42.6±2.3*	485.9±16.0*	3.4±0.6	57.2±1.3*
	E2	35.0±2.5	43.6±2.2*	518.7±31.4*	2.8±0.4	81.2±2.**4****
	E3	36.6±2.5	47.2±2.1*	458.7±45.8	3.8±0.9	46.5±1.**7*****
5th	C	36.4±2.7	48.0±1.8	414.5±14.7	2.6±0.5	60.9±1.5
	E1	40.8±1.2	54.2±1.4*	563.3±24.0*	5.4±0.7*	53.5±0.8*
	E2	37.0±2.0	57.2±2.1*	555.7±25.3*	3.4±0.3	76.0±2.**1****
	E3	41.8±2.4	58.8±1.7*	562.9±46.7*	5.8±1.2*	45.0±0.9*
14th	C	38.2±2.4	48.8±1.6	419.1±22.7	3.4±0.3	57.2±2.1
	E1	38.8±1.2	59.4±1.2*	619.2±20.8*	7.6±0.7*	51.5±0.9*
	E2	50.8±0.9**	60.8±1.2*	578.6±28.4*	5.2±0.7*	64.6±3.5
	E3	58.8±2.7*	69.6±1.2*	589.5±39.5*	7.0±1.0*	44.1±1.**5****
30th	C	37.0±3.0	51.6±2.1	428.6±27.5	4.0±0.3	67.3±3.5
	E1	53.8±1.7**	62.0±1.0*	749.2±27.9*	8.2±0.7*	48.9±1.5*
	E2	59.0±1.3*	69.2±1.6*	659.0±32.7*	5.6±0.3*	60.0±2.9
	E3	61.8±1.2*	75.8±1.9*	658.0±38.4*	7.2±0.9*	45.5±1.**7*****
55th	C	40.4±2.3	48.0±0.8	471.1±21.0	4.6±0.7	72.1±3.5
	E1	60.6±0.8*	46.8±0.7	772.0±32.5*	8.8±0.4*	52.4±2.**7****
	E2	59.0±1.0*	48.2±1.8	730.0±34.0*	6.2±0.4*	62.2±2.7*
	E3	63.8±2.2*	44.2±1.9*	688.0±35.0*	7.2±0.7*	50.8±1.8*

Note: * p≤0.001, ** p≤0.01, *** p≤0.05 compared to the values of the control group of dogs; C - control, E1, E2, E3 - experimental groups

ALT activity in the blood plasma *of the control* group animals deviated slightly from the physiological norm, which may be a consequence of mechanical trauma to the spine and adjacent soft tissues, as well as the development of concomitant hepatopathology.

In dogs *of the first* experimental group, a slight increase in ALT activity was detected on days 1 and 5 of treatment, respectively, by 35.7 and 12.9 % compared with the control. At the same time, on the 14th and 30th day of the experiment, this indicator increased slightly, by 21.7 and 20.2 %, respectively, compared to the control. At the same time, on the 55th day of treatment of experimental dogs, the restoration of this indicator was noted, which indicates the effectiveness of the therapy used.

In dogs *of the second* experimental group, a significant increase in plasma ALT activity was observed on the 1st and 5th day of treatment, respectively, by 19.2 and 38.9 % compared to the control. Subsequently, on the 14th and 30th day of treatment, the activity of the studied enzyme remained elevated, respectively by 24.6 and 34.1 % compared to the control. It should be noted that this indicator reached the values of the control group of animals only on day 55, as in the previous groups.

In dogs *of the third* experimental group, significant changes in ALT activity were detected from day 1 to day 30 of treatment, which corresponded to an increase in enzyme activity by 50.3%, 22.5%, 42.6% and 46.9% compared to the control, and is a clear reflection of the general condition of the animals. And only on the 55th day of treatment, a decrease in plasma ALT activity by 7.9 % was found.

Amylase activity in the blood plasma of dogs of *the control* group was within normal limits during the entire study period (Table 3.24).

During the study, an increase in amylase activity was observed in animals of *the first* experimental group at all stages of treatment, especially pronounced on days 30 and 55. Thus, in these animals, the activity of the enzyme increased by 21.9% on day 1, by 35.9% on day 5, by 47.8% on day 14, by 74.8% on day 30, and by 63.9% on day 55 compared to the control, which may indicate a high metabolic activity of the pancreas in injured animals.

In dogs *of the second* experimental group, when treating spinal cord compression syndrome, a similar trend in changes in plasma amylase activity was observed, but less pronounced. In particular, on the 1st, 5th, 14th, 30th and 55th day of treatment, it increased by 30.2 %, 34.1 %, 38.1 %, 53.8 % and 55.0 %, respectively, compared to the control.

Treatment of dogs *of the third* experimental group, like the previous two, was also accompanied by an increase in the activity of amylase in blood plasma, which was manifested from day 5 of the study. Thus, on day 5 it increased by 35.8%, on day 14 - by 40.7%, on day 30 - by 53.5%, on day 55 - by 46.0% compared to the control, which may be due to the severity of the pathological condition and the reaction of the pancreas to the therapy.

In the experimental animals of *the control* group, the activity of **GGTP** during the study period was within the average normal range, which positively characterises the enzymatic activity of the liver, spleen, and pancreas (see Table 3.24).

In dogs *of the first* experimental group, the activity of GGTP increased on the 5th, 14th, 30th and 55th day of treatment, respectively, by 107.7%, 123.5%, 105.0% and 91.3% compared to the control, which may indicate the presence of cholestasis in these animals, the manifestation of which decreases over time.

At the same time, in dogs *of the second* experimental group, the activity of GGTP in the blood plasma began to increase from the 14th day of treatment to the 55th day inclusive, which, according to the observation stage, changed by 52.9%, 40.0% and 34.8% compared to the control group, indicating a positive trend.

A certain analogy was noted in dogs *of the third* experimental group with animals of the first experimental group, in which on the 5th, 14th, 30th and 55th day of the treatment course, a significant increase in the activity of GGTP was found, respectively, by 123.1%, 105.8, 80.0% and 56.5% compared to the control group. This may be the result of the development of cholestasis and cholangitis in these animals, the intensity of which gradually decreases.

The activity of **alkaline phosphatase** in the blood plasma of dogs of *the control* group fluctuated slightly within the reference range during the study period.

Table 3.25 - Biochemical parameters of blood plasma in experimental dogs (M ± m, n = 5 - 15)

Day of experiment	Group.	Protein, g/l	Urea, mmol/l	Creatinine, µmol/l	Bilirubin, µmol/l	Glucose, mmol/l
		Reference values for norms [133, 134]				
		53-76	2.0-8.3	26-120	0.0-15.3	3.5-7.3
1st	C	54.2±1.6	2.8±0.1	87.1±3.1	3.32±0.40	3.9±1.0
	E1	58.6±0.8*	4.0±0.2**	73.8±3.4*	4.42±0.50	6.5±0.1*
	E2	58.4±3.2	4.0±0.1**	157.8±7.7*	3.14±0.30	4.5±0.1**
	E3	49.6±1.2*	4.4±0.3**	229.8±21.9*	4.20±0.50	6.9±0.1*
5th	C	57.1±1.5	2.9±0.2	92.4±2.5	3.46±0.40	3.7±0.1
	E1	64.7±0.7*	4.4±0.2*	81.6±2.6*	4.48±0.40	6.6±0.2*
	E2	67.8±2.3*	4.4±0.2*	175.6±9.9*	3.52±0.30	4.5±0.1*
	E3	64.5±6.4	7.1±0.5*	250.9±23.1*	4.52±0.60	7.3±0.1*
14th	C	61.1±1.8	3.0±0.1	101.5±3.5	3.52±0.30	4.1±0.2
	E1	69.5±1.2*	4.6±0.1***	91.4±3.4*	4.44±0.40	6.7±1.0*
	E2	74.6±1.1*	4.9±0.1*	244.5±21.0*	4.10±0.50	4.0±0.1
	E3	68.8±5.5	8.4±0.1*	308.8±41.3*	5.52±0.40*	7.3±0.2*
30th	C	62.7±1.9	3.8±1.0	106.5±2.3	3.68±0.30	4.2±0.2
	E1	77.3±1.0*	5.1±0.3*	95.7±3.1*	4.66±0.40*	6.9±1.0*
	E2	76.4±2.6*	6.2±0.2*	222.1±11.0*	4.38±0.50	4.0±0.1
	E3	72.9±4.4*	8.9±0.1*	363.1±44.2*	5.62±0.30*	7.4±0.1*
55th	C	62.5±2.2	4.1±0.2	117.2±6.0	4.42±0.40	4.2±0.2
	E1	69.8±1.3*	5.3±0.3**	107.0±4.8	5.54±0.30*	6.6±0.2*
	E2	67.7±2.1	6.2±0.1*	264.1±23.5*	4.46±0.40	4.3±0.1
	E3	79.5±1.0*	9.1±0.3***	421.3±35.6*	3.32±0.40	7.8±0.1*

Note: * p≤0.001, ** p≤0.01, *** p≤0.05 compared to the values of the control group of dogs; C - control, E1, E2, E3 - experimental groups

When studying the activity of alkaline phosphatase in the blood plasma of experimental dogs of *the first* group, a decrease in its value was noted throughout the study period, which may be an effect of the use of a glucocorticosteroid drug and the

neuroprotectant Armadin. In the animals *of the second* E2 group, the activity of alkaline phosphatase was characterised by an increase on the 1st and 5th day of treatment, which corresponded to an increase of 30.3 and 24.8%, and a gradual decrease in its value on the 55th day of treatment (by 13.7%), compared with the control group, which reflects the clinical condition of the animals of this group.

The third experimental group of dogs was characterised by a decrease in the activity of the enzyme in the blood plasma during the entire treatment period, respectively by 25.4 %, 26.1, 22.9, 32.4 and 29.5 % compared to the control. This fact may be due to the administration of high doses of glucocorticosteroid drugs to sick animals to prevent the development of neurological deficits and tetraparesis.

The results of the study of a complex of biochemical parameters in the blood plasma of dogs of the control and experimental groups during the treatment period are given in Table 3.25.

During the experiment, the total **protein** content of the *control* group animals was at the lower limit of the reference standard, which is explained by the complete and balanced feeding of patients after analgesia as a result of severe pain (see Table 3.25).

In general, under different schemes of complex treatment of animals of the three experimental groups, a slight increase in the content of total protein in the blood plasma was observed, which positively characterises the effect of the therapy.

Thus, in dogs of *the first* experimental group, a slight increase in the content of total protein in the blood plasma was noted at all stages of treatment. In particular, on the 1st, 5th, 14th, 30th and 55th day, this indicator increased by 8.1%, 13.3, 13.7, 23.3 and 11.7%, respectively, compared to the control.

In dogs *of the second* experimental group, a gradual increase in the content of total protein in the blood plasma was found, although not at all stages of treatment. A significant increase in the content of this indicator in the blood plasma of animals was observed on the 5th, 14th and 30th day of treatment, respectively, by 18.7%, 22.1% and 21.9% compared to the control.

In dogs *of the third* experimental group, on the 1st day of treatment with the

development of spinal shock and spinal cord injury, a slight hypoproteinaemia was noted, which corresponds to a decrease in the content of total protein in the blood plasma by 8.5 % compared to the control. And only on the 30th and 55th day of treatment, an increase in the level of total protein in the blood plasma was found, respectively, by 6.0 and 27.2%, which indicates a positive effect of the therapy.

The urea concentration in the blood plasma *of the control* group dogs was within the normal range throughout the study period.

In the dogs of *the first* experimental group, its concentration remained within the reference norm, but increased compared to the control group of animals. Hence, it follows that adequate nutrition and the applied therapeutic regimen had a positive effect on the functional state of the body of dogs with combined spinal cord injury.

In the animals of *the second* experimental group, the plasma urea concentration also did not differ from the reference range, and compared to the first experimental group, it increased in the range from 29.3 to 53.3 % throughout the study period. Despite the development of the ascending syndrome and the gradual increase in neurological deficit, the experimental dogs did not develop renal failure (see Table 3.25).

Dogs *of the third* experimental group showed a significant increase in blood plasma urea concentration from day 14 to day 55 of treatment, by 180.0%, 134.2% and 122.0%, respectively, compared to the control group of animals, which may be due to the prolonged shock state of the injured animals, increased catabolic processes against the background of severe spinal cord injury and significant impairment of the functional state of the kidneys.

In the blood plasma *of the control* group, the **creatinine** concentration was at the level of the average normal limit. It should be noted that on the 55th day of the experiment, this indicator underwent changes that numerically approached the upper limit of normal (see Table 3.25).

During the study period, physiological levels of creatinine were recorded in the blood plasma of dogs of *the first* experimental group, which, at the same time, were characterised by a tendency to decrease in value compared with the values of the

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control group. This situation may be a consequence of impaired creatinine formation at the level of muscle tissue.

On the contrary, in dogs *of the second* experimental group, the creatinine concentration from day 1 to day 55 of the complex treatment (prednisolone + ascorbic acid 5% + butomidor) was characterised by high values that exceeded the control level by 81.7%, 90.0%, 140.1%, 108.5% and 125.3%, respectively, during the observation period, which may be caused by changes in the filtration capacity of the kidneys.

The most difficult group in comparison with the previous ones is *the third* experimental group of dogs, in which the creatinine content in the blood plasma was moderately elevated on the 1st and 5th day of observation, which corresponded to an increase in the value of the indicator by 163.8 and 122.8 % compared to the control. High values of this indicator were detected on the 14th, 30th and 55th day of the treatment course, which was characterised by an increase of 204.2 %, 240.9 % and 259.5 %, respectively. This fact may indicate a violation of the functional state of the kidneys in animals of this group.

The total **bilirubin** content in the blood plasma *of the control* group dogs remained within the reference range throughout the study.

In the dogs of *the first* experimental group, the content of total bilirubin in the blood plasma increased in the last stages of the experiment, namely, on the 30th and 55th day of treatment, respectively, by 26.6 and 25.3% compared to the control, which may be due to the liver's response to intensive care and intoxication of the body with tissue destruction products in spinal cord injury.

In the animals of *the second* experimental group, the level of total bilirubin in the blood plasma remained unchanged throughout the treatment course.

In dogs *of the third* experimental group, the content of total bilirubin in the blood plasma was characterised by an increase on the 14th, 30th and 55th day of treatment, respectively, by 56.8 %, 52.7 and 33.9 % compared to the control. At the same time, during the entire observation period, no changes in the visible mucous membranes and skin were observed in the dogs.

The plasma **glucose** concentration *in the control* group dogs was within the

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average normal range (see Table 3.25).

In the animals of *the first* experimental group, an increase in blood plasma glucose concentration was observed at all stages of observation, which may be the result of the use of appropriate drugs and the body's response to pain.

In dogs *of the second* experimental group, a slight increase in blood plasma glucose concentration was observed only on days 1 and 5 of treatment, respectively, by 15.4 and 21.6 % compared with the control, although the clinical condition of the animals was severe.

Conversely, in animals *of the third* experimental group, starting from the 5th day of treatment, moderate hyperglycaemia was established, which was marked by an increase in blood plasma glucose levels by 97.3 %, 78.0, 76.2 and 85.7 %, respectively, which was clinically manifested by polydipsia. This, in turn, explains the development of a shock state due to the received SCI and is also the result of the use of high doses of glucocorticosteroid drugs.

2.6. Conclusion to Section 2

Postoperative rehabilitation: all sick animals were recommended to be kept in an enclosure or in a room with limited space to prevent sudden movements and free movement. The dogs were kept for 5 to 8 weeks. Sick dogs were treated with cephalosporin and fluoroquinolone antibiotics in therapeutic doses.

Evaluation of treatment effectiveness: 5 dogs of the first experimental group (E1) showed 100% recovery after a comprehensive examination of patients on day 55 during the rehabilitation course and the last blood sample for haematological examination.

In the second experimental group, 60% (3 dogs) had complete loss of pelvic limb movement due to spontaneous development of ascending syndrome from day 35 and 40% (2 dogs) of patients with restoration of locomotor function on day 55 of clinical examination and rehabilitation.

In the animals of the third experimental group, the treatment results were 40 %

(2 dogs) with partial loss of motor function before movement as a result of complex SCI and 60 % (3 dogs) with restoration of motor function during the observation and postoperative rehabilitation period, which is a high result for the thoracolumbar spine injury.

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CONCLUSIONS

The distribution, symptom complex and diagnostic value of complex research methods in various clinical forms of spinal cord and spinal cord injuries in dogs are investigated. The monograph provides theoretical and clinical substantiation of drugs with different pathogenetic effects and methods of surgical intervention in the treatment of dogs with various neurological syndromes.

1. A total of 742 dogs were examined during the study period, of which 155 dogs (20.88%) were diagnosed with Hansen type I and II intervertebral disc disease, spinal cord and spinal cord injuries during clinical examination and additional research methods (orthopaedic, neurological, radiological, tomographic).

2. Out of the 155 dogs (20.88% of their total number) studied, the following neurological syndromes caused by spinal cord injuries were diagnosed

Cauda equina syndrome - 10 (6.45 %), lumbosacral syndrome - 7 (4.51 %), radicular syndrome - 26 (16.77 %), Schiff-Sherrington syndrome - 5 (3.22 %) and ascending syndrome - 5 (3.22 %) cases.

Based on the data obtained, a control group (n = 5) and 3 experimental groups consisting of 15 dogs (E1, E2, E3, n = 5 each) were formed: Cauda equina syndrome, lumbosacral syndrome, root syndrome, Schiff-Sherrington syndrome and ascending syndrome.

3. For dogs of the 1st experimental group (n = 5) with lumbosacral syndrome, surgical methods of treatment were used in the form of hemilaminectomy with foramenectomy and hemilaminectomy with facetectomy, as well as decompression and stabilisation surgery at the level of L7-S1 (seventh lumbar and first sacral) vertebrae for 5 dogs. One animal additionally underwent vertebral body stabilisation surgery, where a fracture of the second vertebra (L2) in the lumbar spine was diagnosed. Postoperative rehabilitation of 4 animals with cauda equina syndrome lasted 30 days. In one dog, the full rehabilitation period was 40 days. As a result, after the neurological examination, there was a complete restoration of the motor apparatus function and no signs of neurological deficit in the pelvic limbs, which amounted to 100% effectiveness of the proposed treatment methods.

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4. For dogs of the 2nd experimental group (n = 5) with spinal cord compression syndrome of the lumbar spine as a result of Hansen type I intervertebral disc disease, which caused the occurrence of root syndrome, surgical methods of treatment in the form of hemilaminectomy in combination with mini-hemilaminectomy were used to form a minor defect in the bone tissue. Spinal cord decompression in 2 animals (40%) led to partial restoration of pelvic limb function and in 3 dogs (60%) the development of ascending syndrome, which caused partial loss of motor activity in the pelvic limbs. The full course of postoperative and physical rehabilitation lasted from 90 to 120 days.

5. For dogs of the 3rd experimental group (n = 5) with Schiff-Sherrington syndrome caused by trauma to the thoracic spine and the development of spinal shock, after normalisation of the condition to physiological parameters, decompression and stabilisation surgery was performed using metal osteosynthesis of vertebral bodies in the thoracic spine. Decompression of the spinal cord in 3 animals (60%) resulted in restoration of pelvic limb function and in 2 dogs (40%) caused partial loss of motor activity of the pelvic limbs due to paresis. The full course of postoperative and physical rehabilitation lasted from 120 to 140 days.

6. For a full neurological examination of experimental dogs, a neurological protocol (Neurological Examination Form (2010)) was used. The data obtained related to neurological disorders and disorders of the musculoskeletal system were recorded in the relevant sections, starting from the moment of initial admission. This, in turn, made it possible to collect data on the examination of the head, cranial nerves, determination of the presence/absence of postural and spinal reflexes, and pain sensitivity. The number of such examinations depended on the severity of the clinical condition of the dogs, and averaged from 2 to 5 protocols during the period of complex treatment and postoperative rehabilitation.

7. High diagnostic efficiency was determined by the method of contrast myelography. In particular, in the control group (n = 5) and experimental groups (E1, E2), there were found disorders of contrast agent columns caused by the formation of Hansen type I and II intervertebral disc herniation and stenosis of the intervertebral space. Dogs of the 3rd experimental group (n = 5) were diagnosed with leakage of

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contrast medium beyond the subarachnoid space caused by trauma to the thoracolumbar spine. As a result, the efficiency of the method was 100%.

8. The most effective diagnostic methods are tomographic (computed tomography, magnetic resonance imaging), which allowed in 100% of cases to confirm the preliminary diagnosis and qualitatively visualise bone and soft tissue after spinal injury.

9. A significant clinical result was obtained from the treatment process in dogs of the first experimental group (E1), which were diagnosed with lumbosacral syndrome. During the conservative treatment, such complexes of drugs as Depo-Medrol (40 mg/ml), Vitaxon and Armadin (50 mg/ml) were used. Surgical treatment included decompression and stabilisation surgery at the level of L7-S1 (seventh lumbar and first sacral) vertebrae. The effectiveness of the treatment course was 100%.

Accordingly, the effectiveness of the treatment process in dogs of the second experimental group (E2) with a rate of 40 % was due to a complication of the spinal cord in the form of the development of a spontaneous ascending syndrome of unknown genesis with a severe neurological deficit.

In the animals of the third group (E3), the effectiveness of the complex treatment process was 60%, which was caused by a spinal cord complication with moderate neurological deficit and damage to the thoracolumbar spine and Schiff-Sherrington syndrome.

10. Physicochemical, cytological and biochemical studies of the cerebrospinal fluid at an early stage allow to identify changes indicative of acute or chronic inflammatory processes of various origins, establishing signs of subarachnoid haemorrhage, myelitis, etc. At the later stages of laboratory tests, signs of xanthochromia and neutrophilic pleocytosis are detected, which indicates polyneuritis with significant cerebrospinal fluid disorders due to a spinal cord injury.

 ISBN 978-3-98924-091-9

LIST OF REFERENCES

1. Lumbar Disc Herniation: Diagnosis and Management. / Zhang A.S., Xu A., Ansari K., Hardacker K., Anderson G., Alsoof D., Daniels A.H. // Am J Med. 2023 Jul;136(7):645-651. doi: 10.1016/j.amjmed.2023.03.024.

2. Dog faces exhibit anatomical differences in comparison to other domestic animals. / Burrows A.M., Kaminski J., Waller B.M., Omstead K.M., Rogers-Vizena C., Mendelson B. // Anat Rec (Hoboken). 2021 Jan;304(1):231-241. doi: 10.1002/ar.24507.

3. Tavee J. Nerve conduction studies: Basic concepts. // Handb Clin Neurol. 2019;160:217-224. doi: 10.1016/B978-0-444-64032-1.00014-X.

4. Wohlsein J.C., Tipold A. Steroid-responsive meningitis-arteritis: What have we learned since 2010? A narrative review. // Vet J. 2023 Oct-Dec;300-302:106030. doi: 10.1016/j.tvjl.2023.106030..

5. 5. Cambau E., Poljak M. Sniffing animals as a diagnostic tool in infectious diseases.// Clin Microbiol Infect. 2020 Apr;26(4):431-435. doi: 10.1016/j.cmi.2019.10.036.

6. MRI features of canine hemangiosarcoma affecting the central nervous system. / Mallol C., Gutierrez-Quintana R., Hammond G., Schweizer-Gorgas D., De Decker S., Novellas R., Espada Y., Oevermann A., Ortega M., Parry A., Coelho A.M., Gonçalves R., Stalin C., Brocal J. // Vet Radiol Ultrasound. 2022 Mar;63(2):185-196. doi: 10.1111/vru.13041.

7. Clinical and magnetic resonance imaging features of lymphoma involving the nervous system in cats. / Durand A., Keenihan E., Schweizer D., Maiolini A., Guevar J., Oevermann A., Gutierrez-Quintana R. // J Vet Intern Med. 2022 Mar;36(2):679-693. doi: 10.1111/jvim.16350.

8. MRI and CT features of a disc herniation through a fractured lumbar vertebra causing lumbar spinal cord compression in a dog. / Capasso M., Dell'Era E., Carloni A., Balducci F., Specchi S., Bernardini M. // Vet Radiol Ultrasound. 2023 Nov;64(6):E64-E67. doi: 10.1111/vru.13284.

9. Algorithm of neurological examination using neurosurgical scales in case of spinal injuries in dogs. Scientific and Technical Bulletin of the State Research and Control Institute of Veterinary Preparations and Feed Additives and the Institute of Animal Biology. 2018. Issue 19, No. 2. P. 92-98.

10. Biloshytskyi R. V. Analysis of a clinical case of Schiff-Sherrington syndrome in a German Shepherd, methods of diagnosis and treatment. Conference materials: The role of innovation in transforming the image of modern science: Proceedings of the II International Scientific and Practical Conference (Kyiv, 28-29 December 2018) / NGO "Institute of Innovative Education"; Scientific and Educational Centre for Applied Informatics of the National Academy of Sciences of Ukraine - Kyiv: NGO "Institute of Innovative Education", 2018. - P. 223-226.

11. Diagnosis of Cauda Equina syndrome in different breeds of dogs. Scientific Bulletin of the National University of Life and Environmental Sciences of Ukraine. Series: Veterinary medicine, quality and safety of livestock products. 2018. Issue 285. P. 27-33.

12. Biloshytskyi R. V. Diagnosis of Cauda Equina syndrome in dogs of brachiocephalic and dolichocephalic type. Conference materials: Science, technology, innovation: global trends and regional aspect: Proceedings of the International Scientific and Practical Conference (Odesa, 28-29 September 2018) / NGO "Institute of Innovative Education"; Scientific and Educational Centre for Applied Informatics of the National Academy of Sciences of Ukraine - Odesa: NGO "Institute of Innovative Education", 2018. P. 185-188.

13. Biloshytskyi R. V. General characteristics of the use of glucocorticosteroid drugs in spinal cord and spinal cord injuries. Conference materials: Modern methods of diagnosis, treatment and prevention in veterinary medicine, Lviv, 29-30 November 2018: abstracts. Lviv, 2018. P. 14-15.

14. Biloshytskyi R. V. Clinical case of Schiff-Sherrington syndrome in a German Shepherd Dog, diagnosis and treatment. Scientific Bulletin of the National University of Life and Environmental Sciences of Ukraine. Series: Veterinary medicine, quality and safety of livestock products. 2018. Issue 293. P. 7-13.

15. Biloshytskyi R.V. Methods of diagnosis and provision of conservative and surgical treatment for discopathies in dogs. *Biological resources and nature management.* Vol. 10, No. 5-6, 2018. P. 189-197.

16. Biloshytskyi R.V. Methods of diagnosis and treatment of ascending syndrome in dogs. Scientific and Technical Bulletin of the Research Centre for Biosafety and Environmental Control of Agricultural Resources: Theoretical and Applied Veterinary Medicine. Vol. 6, No. 3 (2018). P. 29-33.

17. Biloshytskyi R. V. Methods of neurological examination using neurosurgical scales in spinal cord injuries in different dog breeds. Actual aspects of animal biology, veterinary medicine and veterinary and sanitary expertise: materials of the III International scientific and practical conference of teachers and students, Dnipro, 16-18 May 2018: abstracts. Dnipro, 2018. P. 17-19.

18. Biloshytskyi R. V. Lumbosacral syndrome in dogs and methods of diagnosis. Animal Biology. 2018. Vol. 20, № 4. P. 87.

19. Biloshytskyi R. V. Practical use of the high-frequency welding electrocoagulator EKVZ-300 "PATONMED" for injuries of the thoracolumbar spine in dogs. Welding and heat treatment of living tissues. Theory. Practice. Prospects: materials of the XIV Scientific and Practical Conference of the E.O. Paton Institute of Electricity of the National Academy of Sciences of Ukraine, Kyiv, 29 November 2019, abstracts. K., 2019. C. 6-7.

20. Practical experience of myelography application in neurological syndromes in dogs. Conference materials: International scientific and practical conference "Food safety control. Ukraine-EU: unresolved issues", Kyiv, 19-20 April 2018: abstracts. K., 2018. C. 128-129.

21. Biloshitsky R. V. Special methods of diagnosis and prescription of surgical treatment for discopathies in dogs. Conference materials: Integration of world scientific processes as the basis of social progress: Proceedings of the II International Scientific and Practical Conference (Kyiv, 23-24 November 2018) / NGO "Institute of Innovative Education"; Scientific and Educational Centre of Applied Informatics of the National Academy of Sciences of Ukraine - Kyiv: NGO "Institute of Innovative

Education", 2018. - pp. 217-221.

22. Biloshytskyi R.V., Sukhonos V.P. Diagnostic value of cerebrospinal fluid examination in dogs with neurological syndromes. Scientific Bulletin of the National University of Life and Environmental Sciences of Ukraine. Series: Veterinary medicine, quality and safety of livestock products. 2017. Issue 265. C. 17-26. 23. Biloshytskyi R.V., Sukhonos V.P. Experience of myelography application in neurological syndromes in dogs. Scientific Bulletin of the National University of Life and Environmental Sciences of Ukraine. Series: Veterinary medicine, quality and safety of livestock products. 2017. Issue 273. C. 231-241.

24. Biloshytskyi R.V., Sukhonos V.P. Experience of surgical treatment of dogs with intervertebral disc dislocations. *Biological resources and nature management.* Vol. 10, No. 3-4, 2018. C. 221-227.

25. Biloshitsky RV, Sukhonos VP, Study of cerebrospinal fluid in dogs with neurological syndromes. Congress materials: Fifteenth International Congress of Veterinary Medicine Specialists, Kyiv, 5-6 October 2017: abstracts. K., 2017. C. 21-24.

26. Biloshitsky RV, Sukhonos VP Clinical experience of surgical treatment of dogs with intervertebral disc dislocations. Congress materials: Sixteenth International Congress of Veterinary Medicine Specialists, Kyiv. Kyiv, 16 October 2018: abstracts. K., 2018. C. 13-15.

27. Biloshitsky RV, Sukhonos VP Complex treatment of dogs with regard to the peculiarities of pathogenesis in spinal cord injuries. Conference materials: Actual problems of science development in the context of global transformations of the information society: Proceedings of the International Scientific and Practical Conference (Kyiv, 26-27 October 2018) / NGO "Institute of Innovative Education"; Scientific and Educational Centre for Applied Informatics of the National Academy of Sciences of Ukraine - Kyiv: NGO "Institute of Innovative Education", 2018. - pp. 163-167.

28. Biloshytskyi R.V., Sukhonos V.P. Treatment of dogs taking into account the peculiarities of the pathogenesis of spinal cord injuries. Scientific reports of the

National University of Life and Environmental Sciences of Ukraine. 2018. № 4 (74).

29. Biloshytskyi R.V., Tarnavskyi D.V. Experience of using a high-frequency welding electrocoagulator in veterinary vertebrology. Welding and heat treatment of living tissues. Theory. Practice. Prospects: materials of the XIII Scientific and Practical Conference of the IES named after - 1 December 2018, Abstracts. K., 2018. P. 12.

30. Biloshytskyi R. V., Tarnavskyi D. V., Tkachenko V. V. Experience of using an electric welding machine during operations after damage to the axial skeleton in dogs. Welding and heat treatment of living tissues. Theory. Practice. Prospects: materials of the XII Scientific and Practical Conference of the E.O. Paton Institute of Electric Welding of the National Academy of Sciences of Ukraine, Kyiv, 1-2 December 2017, Abstracts. K., 2017. P. 24.

31. Closure times of neurocranial sutures and synchondroses in Persian compared to Domestic Shorthair cats. / Schmidt M.J., Farke D., Staszyk C., Lang A., Büttner K., Plendl J., Kampschulte M. // Sci Rep. 2022 Jan 12;12(1):573. doi: 10.1038/s41598-022-04783-1.

32. Significant Neuroanatomical Variation Among Domestic Dog Breeds. / Hecht E.E., Smaers J.B., Dunn W.D., Kent M., Preuss T.M., Gutman D.A. // J Neurosci. 2019 Sep 25;39(39):7748-7758. doi: 10.1523/JNEUROSCI.0303-19.2019.

33. Vyshniak O. S. Diagnostics and conservative treatment of spinal diseases in dogs and cats: thesis for the degree of Candidate of Veterinary Sciences in the speciality 16.00.05 "Veterinary Surgery". Kyiv, 2007. P. 10-136.

34. Seeram E. Computed Tomography: A Technical Review. // Radiol Technol. 2018 Jan;89(3):279CT-302CT

35. A computed tomography-based method for the assessment of canine retrobulbar cone volume for ophthalmic anaesthesia. / Greco A., Costanza D., Senatore A., Bruzzese D., Micieli F., Chiavaccini L., Di Giancamillo M., Della Valle G., Vesce G., Brunetti A., Meomartino L. // Vet Anaesth Analg. 2021 Sep;48(5):759-766. doi: 10.1016/j.vaa.2021.03.015.

36. Rossetti D, Ragetly GR, Poncet CM. High-Definition Video Telescope-

Assisted Ventral Slot Decompression Surgery for Cervical Intervertebral Disc Herniation in 30 Dogs.// Vet Surg. 2016 Oct;45(7):893-900. doi: 10.1111/vsu.12528.

37. Moore P.F. A review of histiocytic diseases of dogs and cats.// Vet Pathol. 2014 Jan;51(1):167-84. doi: 10.1177/0300985813510413.

38. Goralskyi LP, Khomych VT, Sokulskyi IM et al. Morphology of the spinal cord and spinal nodes of vertebrates: [monograph]. Lviv: ZUKC, 2016. P. 99-105.

39. Goralsky L.P., Khomych V.T., Shikh Y.S., Dekhtyarev P.A., Samoilyuk V.V. Anatomy and features of dog physiology with the basics of training: a manual. Zhytomyr: Polissya, 2008. P. 274-323.

40. . Signalment, clinical presentation, and diagnostic findings in 122 dogs with spinal arachnoid diverticula. / Mauler D.A., De Decker S., De Risio L., Volk H.A., Dennis R., Gielen I., Van der Vekens E., Goethals K., Van Ham L.// J Vet Intern Med. 2014 Jan-Feb;28(1):175-81. doi: 10.1111/jvim.12241.

41. Arzi B. Contemporary management of temporomandibular joint fractures in dogs and cats: review and expert insights on diagnostic imaging, treatment strategies, and long-term outcomes. // J Am Vet Med Assoc. 2023 Jun 19;261(S2):S44-S51. doi: 10.2460/javma.23.04.0211.

42. Cytological and histological correlation in diagnosing feline and canine mediastinal masses. / Pintore L., Bertazzolo W., Bonfanti U., Gelain M.E., Bottero E. // J Small Anim Pract. 2014 Jan;55(1):28-32. doi: 10.1111/jsap.12161.

43. Menghini T.L., Shriwise G., Muir P. Fracture Healing in 37 Dogs and Cats with Implant Failure after Surgery (2013-2018). // Animals (Basel). 2023 May 5;13(9):1549. doi: 10.3390/ani13091549.

44. Anatomy, Back, Cauda Equina / E.J. Berg, J.V. Ashurst // In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. 2023 Jul 25. (https://pubmed.ncbi.nlm.nih.gov/30020623/).

45. Zozulya Y.A., Pedachenko E.G., Slynko E.I. Surgical treatment of neurocompression lumbosacral pain syndromes. K.: UIPK Exob. 2006. P. 21-81.

46. Ivanov V.O., Kostiuk V.K., Samoiliuk V.V. Morphology of farm animals (in diagrams): a manual. Kherson: Aldi-plus, 2012. P. 162-175.

47. Global Neurosurgery: An Overview. / Garcia R.M., Shlobin N.A., Baticulon R.E., Ghotme K.A., Lippa L., Borba L.A., Qureshi M., Thango N., Khan T., Hutchinson P., Rosseau G. // Neurosurgery. 2024 Sep 1;95(3):501-508. doi: 10.1227/neu.0000000000003109.

48. Towards mHealth applications for pet animal owners: a comprehensive literature review of requirements. / Haase L., Sedlmayr B., Sedlmayr M., Monett D., Winter J. // BMC Vet Res. 2025 Mar 21;21(1):190. doi: 10.1186/s12917-025-04658-3.

49. Microendoscopic Mini-Hemilaminectomy and Discectomy in Acute Thoracolumbar Disc Extrusion Dogs: A Pilot Study. / Kamishina H., Nakano Y., Nozue Y., Nakata K., Kimura S., Drury A.G., Maeda S. // Vet Sci. 2021 Oct 18;8(10):241. doi: 10.3390/vetsci8100241.

50. Canine Cognitive Dysfunction: Pathophysiology, Diagnosis, and Treatment. / Dewey C.W., Davies E.S., Xie H., Wakshlag J.J. // Vet Clin North Am Small Anim Pract. 2019 May;49(3):477-499. doi: 10.1016/j.cvsm.2019.01.013.

51. Dewey C.W. Anticonvulsant therapy in dogs and cats. \\ Vet Clin North Am Small Anim Pract. 2006 Sep;36(5):1107-27, vii. doi: 10.1016/j.cvsm.2006.05.005.

52. Dewey C.W, Brunke M.W, Sakovitch K. Transcranial photobiomodulation (laser) therapy for cognitive impairment: A review of molecular mechanisms and potential application to canine cognitive dysfunction (CCD). // Open Vet J. 2022 Mar-Apr;12(2):256-263. doi: 10.5455/OVJ.2022.v12.i2.14.

53. Kemelman E.L. X-ray morphological features of pathological changes of the spinal column in dogs at computed tomography: thesis for the degree of Candidate of Veterinary Sciences in the speciality 16.00.02 "Pathology, Oncology and Morphology of Animals". M., 2013. P. 99.

54. Kemelman E. L., Shchurov I. V., Vatnikov Y. A. Diagnostics of herniated intervertebral discs in chondrodystrophic dog breeds using computed tomography. Actual issues of veterinary biology. 2017. №2 (14). P. 58 - 63.

55. Kononsky O. I. Biochemistry of animals: a textbook. K.: Higher School, 2006. P. 328-335.

56. Intra-abdominal blockage of a ventriculoperitoneal shunt by a suspected

mesenteric pseudocyst in a dog. / Driver C.J., Pinilla M.J., Loderstedt S., Kenny P.J. // Aust Vet J. 2013 Oct;91(10):418-421. doi: 10.1111/avj.12101.

57. Clinical features, comparative imaging findings, treatment, and outcome in dogs with discospondylitis: A multi-institutional retrospective study. / Van Hoof C., Davis N.A., Carrera-Justiz S., Kahn A.D., De Decker S., Grapes N.J., Beasley M., Du J., Pancotto T.E., Suñol A., Shinn R., DeCicco B., Burkland E., Cridge H. // J Vet Intern Med. 2023 Jul-Aug;37(4):1438-1446. doi: 10.1111/jvim.16785.

58. Thoracic Wall Reconstruction with Dorsal Diaphragmatic Traction and Preservation of Diaphragmatic Attachments in a Dog with Resection of the 11-13th Ribs. / Kanai E., Kudo A., Sato A., Yoshida H., Yamauchi A., Oshita R., Takagi S. // Animals (Basel). 2022 Dec 21;13(1):34. doi: 10.3390/ani13010034.

59. International veterinary canine dyskinesia task force ECVN consensus statement: Terminology and classification. / Cerda-Gonzalez S., Packer R.A., Garosi L., Lowrie M., Mandigers P.J.J., O'Brien D.P., Volk H.A. // J Vet Intern Med. 2021 May;35(3):1218-1230. doi: 10.1111/jvim.16108.

60. Chrisman S, Mariani K, Platt S, Clemons R. Neurology of canine cats. A reference guide for practising veterinarians. M: Aquarium Print, 2016. P. 271-427.

61. Lokes PI, Stovba VG, Kravchenko SO, Karisheva LP, Grishchuk AV Computer, magnetic resonance imaging and other modern diagnostic methods in veterinary medicine of small animals: a manual. Poltava: Environment-K, 2011. P. 40-92.

62. An internet survey of the perceived prevalence of zoological companion animal (ZCA) species being evaluated by ACVECC diplomates and emergency and critical care residents and to gauge potential interest in advanced training opportunities in the emergency treatment of these patients. / Sands J., Cudney S., Graham J., Bercier M., Mayer C., Rozanski E., Schoeffler G. // J Vet Emerg Crit Care (San Antonio). 2022 Nov;32(6):830-835. doi: 10.1111/vec.13223.

63. von Dehn B. Pediatric clinical pathology. // Vet Clin North Am Small Anim Pract. 2014 Mar;44(2):205-19. doi: 10.1016/j.cvsm.2013.10.003.

64. Meyer D.J., Harvey J.W. Hematologic changes associated with serum and

hepatic iron alterations in dogs with congenital portosystemic vascular anomalies. // J Vet Intern Med. 1994 Jan-Feb;8(1):55-6. doi: 10.1111/j.1939-1676.1994.tb03196.x.

65. Investigational Therapies for Gunshot Wounds to the Spine: A Narrative Review. / Somuncu D., Gartenberg A., Cho W. // Clin Spine Surg. 2022 Jul 1;35(6):233-240. doi: 10.1097/BSD.0000000000001258.

66. Miroshnychenko S. Digital receivers of X-ray images. K.: Medicine of Ukraine, 2014. P. 11-21.

67. Thrall D.E., Robertson I.D. Atlas of Normal Radiographic Anatomy and Anatomic Variants in the Dog and Cat, 3rd Edition. 2022.

68. Potential treatment targets for migraine: emerging options and future prospects./ Chiang C.C., Porreca F., Robertson C.E., Dodick D.W. // Lancet Neurol. 2024 Mar;23(3):313-324. doi: 10.1016/S1474-4422(24)00003-6.

69. Antończyk A, Ochota M. Is an epidural component during general anaesthesia for caesarean section beneficial for neonatal puppies' health and vitality? // Theriogenology. 2022 Jul 15;187:1-8. doi: 10.1016/j.theriogenology.2022.04.015.

70. Adami C., Gendron K. What is the evidence? The issue of verifying correct needle position during epidural anaesthesia in dogs. // Vet Anaesth Analg. 2017 Mar;44(2):212-218. doi: 10.1016/j.vaa.2016.03.003.

71. Nesterova S.V. Epidural anaesthesia in dogs and cats. *The world of veterinary medicine.* 2014. № 4-5 (20). P. 20-24.

72. Ahmad P., Siqueira W.L. Mass spectrometry-based proteomics profiling of dogs with and without oral diseases: a systematic review. // BMC Oral Health. 2024 Mar 22;24(1):369. doi: 10.1186/s12903-024-04096-x.

73. Novak V.P., Bychkov Y.P., Pylypenko M.Y. Cytology, histology, embryology: a textbook. K.: Dakor, 2008. P. 179-226.

74. Thoracolumbar meningeal fibrosis in pugs. / Rohdin C., Ljungvall I., Häggström J., Leijon A., Lindblad-Toh K., Matiasek K., Rosati M., Wohlsein P., Jäderlund K.H. // J Vet Intern Med. 2020 Mar;34(2):797-807. doi: 10.1111/jvim.15716.

75. RANO 2.0: critical updates and practical considerations for radiological assessment in neuro-oncology. / Sakata A., Fushimi Y., Oshima S., Uto M., Mineharu

Y., Nakajima S., Okuchi S., Yamamoto T., Otani S., Ikeda S., Takada S., Mizowaki T., Arakawa Y., Nakamoto Y. // Jpn J Radiol. 2025 Jun 30. doi: 10.1007/s11604-025-01821-6.

76. Prevalence, distribution, and clinical associations of suspected postictal changes on brain magnetic resonance imaging in epileptic dogs. / Maeso C., Sánchez-Masian D., Ródenas S., Font C., Morales C., Domínguez E., Puig J., Arévalo-Serrano J., Montoliu P. // J Am Vet Med Assoc. 2021 Nov 18;260(1):71-81. doi: 10.2460/javma.21.02.0088.

77. Paton BE, Ivanova ON, et al. Tissue-preserving high-frequency electric welding surgery. Atlas. K.: Scientific and Production Enterprise Publishing House "Naukova Dumka" of the National Academy of Sciences of Ukraine, 2009. P. 100-129.

78. Pedachenko E.G., Polishchuk M.E., Slynko E.I., Khyzhnyak M.V., Pedachenko Y.E., Honda O.M. Traumatic injuries of the spine and spinal cord: [monograph]. K.: Interservice, 2017. P. 6-306.

79. Polatayko O. Veterinary anaesthesia: a practical guide. K.: Periscope Publishing House, 2009. P. 297-300.

80. Poluliakh M.V., Gerasimenko S.I., Skliarenko E.T. et al. Diagnosis and treatment of ankylosing spondylitis. K., 2010. P. 48-105.

81. Portugees A. A., Beletsky E. V., Yusipov V. V. Atlantoaxial instability. A modern view of the problem. The *world of veterinary medicine.* 2017. № 3 (36). P. 20-25.

82. Effects of perineural administration of ropivacaine combined with perineural or intravenous administration of dexmedetomidine for sciatic and saphenous nerve blocks in dogs. / Marolf V., Ida K.K., Siluk D., Struck-Lewicka W., Markuszewski M.J., Sandersen C. // Am J Vet Res. 2021 Jun;82(6):449-458. doi: 10.2460/ajvr.82.6.449.

83. Slynko I.E., Honda A.N. Traumatic injuries of the spine and spinal cord: [monograph]. K.: PP Gama-Print, 2010. P. 172-288.

84. An update on the pathogenesis of syringomyelia secondary to Chiari-like malformations in dogs. / Driver C.J., Volk H.A., Rusbridge C., Van Ham L.M. // Vet J.

2013 Dec;198(3):551-9. doi: 10.1016/j.tvjl.2013.07.014.

85. Kube S.A., Olby N.J. Managing acute spinal cord injuries. // Compend Contin Educ Vet. 2008 Sep;30(9):496-504; quiz 504, 506.

86. Feuer H. Management of acute spine and spinal cord injuries. Old and new concepts. // Arch Surg. 1976 Jun;111(6):638-45. doi: 10.1001/archsurg.1976.01360240018003.

87. Prospective randomized trial comparing relapse rates in dogs with steroid-responsive meningitis-arteritis treated with a 6-week or 6-month prednisolone protocol. / Rose J.H., Driver C.J., Arrol L., Cardy T.J.A., Tabanez J., Tauro A., Fernandes R., Schofield I., Adamantos S., Granger N., Harcourt-Brown T.R. // J Vet Intern Med. 2024 Jul-Aug;38(4):2221-2227. doi: 10.1111/jvim.17130.

88. Wohlsein J.C., Tipold A. Steroid-responsive meningitis-arteritis: What have we learned since 2010? A narrative review. // Vet J. 2023 Oct-Dec;300-302:106030. doi: 10.1016/j.tvjl.2023.106030.

89. Biomarkers of non-infectious inflammatory CNS diseases in dogs: Where are we now? Part 2 - Steroid responsive meningitis-arteritis. / Andersen-Ranberg E., Berendt M., Gredal H. // Vet J. 2021 Jul;273:105692. doi: 10.1016/j.tvjl.2021.105692.

90. Protozoal meningoencephalitis and myelitis in 4 dogs associated with Trypanosoma cruzi infection. / Landsgaard K.A., Milliron S.M., Faccin M., Broughton C.A., Auckland L.D., Edwards J.F., Hamer S.A., Hensel M.E. // Vet Pathol. 2023 Mar;60(2):199-202. doi: 10.1177/03009858221148510.

91. Clinical, imaging, and pathologic characteristics of Gurltia paralysans myelopathy in domestic cats from Chile. / Mieres M., Gómez M.A., Lillo C., Rojas M.A., Moroni M., Muñoz P., Acosta-Jamett G., Wiegand R. // Vet Radiol Ultrasound. 2013 May-Jun;54(3):237-44. doi: 10.1111/vru.12026.

92. Periosteal osteosarcoma in the long head of the triceps in a dog / Cook J.L., Huss B.T., Johnson G.C. // J Am Anim Hosp Assoc. 1995 Jul-Aug;31(4):317-20. doi: 10.5326/15473317-31-4-317.

93. Clinical presentation, diagnostic findings, prognostic factors, treatment and outcome in dogs with meningoencephalomyelitis of unknown origin: A review. /

Cornelis I., Van Ham L., Gielen I., De Decker S., Bhatti S.F.M. // Vet J. 2019 Feb;244:37-44. doi: 10.1016/j.tvjl.2018.12.007.

94. Prolonged Visual Evoked Potential Latencies in Dogs Naturally Infected with Canine Distemper Virus. / Gutiérrez M., Delucchi L., Bielli A., Verdes J.M. // Viruses. 2024 Oct 31;16(11):1721. doi: 10.3390/v16111721.

95. Lumbosacral transitional vertebrae in small-breed dogs: Prevalence, classification, and characteristics. / Kim U., Chang J., Yoon K., Kim B., Ban J., Hyun S.H., Chang D., Lee N. // Vet Radiol Ultrasound. 2025 Jan;66(1):e13469. doi: 10.1111/vru.13469.

96. Congenital and acquired anomalies of the caudal vertebrae in dogs: Radiographic classification and prevalence evaluation. / Paninárová M., Stehlík L., Proks P., Vignoli M. // Acta Vet Hung. 2016 Sep;64(3):330-339. doi: 10.1556/004.2016.031..

97. Sukhonos V.P., Biloshytskyi R.V. Spinal injuries in dogs (clinical forms of spinal cord injuries, diagnosis and treatment): [scientific and practical recommendations]. K., 2019. 46 p.

98. Sukhonos VP, Malyuk MO, Kulida MA, Solonin PK, Doroshchuk VO, Tkachenko SM, Tkachenko VV Orthopedics of dogs and cats. Surgical pathology of the skull and spine. K.: CP Print, 2018. P. 21-103.

99. Sukhonos VP, Malyuk MO, Kulida MA, Tkachenko SM Orthopedics of dogs and cats. Orthopaedic surgical pathology. K. : CP Print, 2017. P. 130-134.

100. Tarnavskyi D.V., Tkachenko V.V., Tkachenko T.A., Biloshytskyi R.V. Experience of using and prospects of the technology of electric welding of living tissues in veterinary surgery. Welding and heat treatment of living tissues. Theory. Practice. Prospects: materials of the XIII Scientific and Practical Conference of the E.O. Paton Institute of Electricity of the National Academy of Sciences of Ukraine, Kyiv, 30 November - 1 December 2018, abstracts. K., 2018. P. 94-99.

101. Application of Shark Teeth-Derived Bioapatites as a Bone Substitute in Veterinary Orthopedics. Preliminary Clinical Trial in Dogs and Cats. / García-González M., Muñoz Guzón F.M., González-Cantalapiedra A., González-Fernández P.M., Otero

Pérez R., Serra Rodríguez J.A. // Front Vet Sci. 2020 Oct 28;7:574017. doi: 10.3389/fvets.2020.574017. eCollection 2020.

102. The standardized creation of a lumbar spine vertebral compression fracture in a sheep osteoporosis model induced by ovariectomy, corticosteroid therapy and calcium/phosphorus/vitamin D-deficient diet. / Eschler A., Röpenack P., Herlyn P.K., Roesner J, Pille K., Büsing K, Vollmar B., Mittlmeier T., Gradl G. // Injury. 2015 Oct;46 Suppl 4:S17-23. doi: 10.1016/S0020-1383(15)30014-0.

103. Clinical and magnetic resonance imaging features of inflammatory versus neoplastic medial retropharyngeal lymph mode mass lesions in dogs and cats. / Johnson PJ, Elders R, Pey P, Dennis R. // Vet Radiol Ultrasound. 2016 Jan;57(1):24-32. doi: 10.1111/vru.12288.

104. Proteomics identifies novel biomarkers of synovial joint disease in a canine model of mucopolysaccharidosis I / Zhang C., Gawri R., Lau Y.K., Spruce LA., Fazelinia H., Jiang Z., Jo S.Y., Scanzello C.R., Mai W., Dodge G.R., Casal M.L., Smith L.J. // Mol Genet Metab. 2023 Feb;138(2):107371. doi: 10.1016/j.ymgme.2023.107371.

105. Hammond L.J., Hecht S. Susceptibility artifacts on T2*-weighted magnetic resonance imaging of the canine and feline spine // Vet Radiol Ultrasound. 2015 Jul-Aug;56(4):398-406. doi: 10.1111/vru.12245.

106. Doroshchuk V.O., Sokol A.I. A clinical case of the treatment of the spine trauma with spinal cord injury in cat. Ukrainian Journal of Veterinary Sciences, 2021. 12(1): 32–39, https://doi.org/10.31548/ujvs2021.01.004

107. Skrypka, M., Panikar, I., Boyko, Yu., Dmytrenko, N., & Kurales, O. (2023). Pathogenesis and pathomorphology of distraction trauma in the framework of pre-trial investigations of cruelty to animals. Scientific Horizons, 26(4), 54-64, DOI: 10.48077/scihor4.2023.54

108. Nazarenko, A.V., Belik, Y.V. (1997). Development of Neurochemical Research in the Ukraine. In: Teelken, A., Korf, J. (eds) Neurochemistry. Springer, Boston, MA. https://doi.org/10.1007/978-1-4615-5405-9_2

109. Khomych V.T., Goralsky L.P., Shikh Yu.S., Morphology of the dog.

Textbook. – Zhytomyr: "Polissya", 2013. 473 p.

110. Thoracic Discogenic Syndrome Delvise T. Fogwe; Brandon Petrone; Sunil Munakomi; Fassil B. Mesfin. Author Information and Affiliations Last Update: March 20, 2024 (https://www.ncbi.nlm.nih.gov/books/NBK470388/)

111. Israel S.K , Levine J.M., Kerwin S.C., Levine G.J., Fosgate G.T. The relative sensitivity of computed tomography and myelography for identification of thoracolumbar intervertebral disk herniations in dogs. Vet Radiol Ultrasound. 2009 May-Jun;50(3):247-52. doi: 10.1111/j.1740-8261.2009.01528.x.

112. Lamb C.R., David F.H. Advanced imaging: use and misuse. J Feline Med Surg. 2012 Jul;14(7):483-97. doi: 10.1177/1098612X12451550.

113. Scientific substantiation of complex diagnostics of spinal neurological syndromes in dogs: author. dissertation. . candidate of veterinary sciences: 16.00.01 "Diagnostics and therapy of animals" (veterinary sciences) / R.V. Biloshitsky; National University of Life Resources and Environmental Sciences. - Kyiv, 2021. - 26 p.

114. Šulla I., BalikV., Horňák S., Ledecký V. Spinal cord injuries in dogs. Part I: a review of basic knowledge. Folia Veterinaria. 2018. 62, 2: 35—44, DOI: 10.2478/fv-2018-0015

115. Smith C.J., Guevar J. Spinal subarachnoid diverticula in dogs: A review. Can Vet J 2020; 61:1162–1169.

116. Treatment of Chronic Spinal Cord Injury in Dogs Using Amniotic Membrane-Derived Stem Cells: Preliminary Results / Orlandin J.R. et al. Stem Cells and Cloning: Advances and Applications. 2021:14 39–49.

117. Bogdan M. Neurological Disorders in Dogs. https://www.petmd.com/dog/conditions/neurological/neurological-disorders-dogs

118. Rusbridge C. Canine chondrodystrophic intervertebral disc disease (Hansen type I disc disease). Rusbridge BMC Musculoskeletal Disorders 2015, 16 (Suppl 1): S11 http://www.biomedcentral.com/1471-2474/16/S1/S11

119. https://www.vet.cornell.edu/departments-centers-and-institutes/riney-canine-health-center/canine-health-information/intervertebral-disc-disease

120. Kealy J.K., Graham J.P. Diagnostic Radiology and Ultrasonography of the

Dog and Cat (Fifth Edition) 2011, https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/spondylitis

121. Jones R., Cirovic S., RusbridgeC. A review of cerebrospinal fluid circulation with respect to Chiari-like malformation and syringomyelia in brachycephalic dogs. Fluids Barriers CNS. 2025 Mar 10;22:25. doi: 10.1186/s12987-025-00636-x

122. Crisp M.J., Beckett J., Coates J.R., Miller T. M. Canine degenerative myelopathy: Biochemical characterization of superoxide dismutase 1 in the first naturally occurring non-human amyotrophic lateral sclerosis model.Experimental Neurology. 2013. Vol. 248. P. 1-9, https://doi.org/10.1016/j.expneurol.2013.05.009

123. https://eu.united-imaging.com/en/news-center/knowledge-base/category-mr/article-70

124. Biloshytskyy, R. V. Methods of diagnosis and treatmentof ascending syndrome in dogs. Theoretical and Applied Veterinary Medicine, 2018. 6(3), 29–33.doi: 10.32819/2018.63006

125. Paithanpagare Y. M. , Tank P. H., Mankad M. Y., Shirodkar K., Derashri H. J. Myelography in dogs. Veterinary World, 2008.Vol.1(5):152-154

126. Butterfield S. Inflammatory diseases of the CNS in dogs. Veterinary Ireland Journal I Vol. 12 No 4, P.203-207.

127 Nicholls M. Sir Godfrey Newbold Hounsfield and Allan M. Cormack. European Heart Journal, Volume 40, Issue 26, 7 July 2019, Pages 2101–2103, https://doi.org/10.1093/eurheartj/ehz421

128. Villagrán J.M., Martínez-Sánchez R T., Cebada-Chaparro E., Horcajadas A L B., Pérez-Fernández E. Diagnostic accuracy of lumbar CT and MRI in the evaluation of chronic low back pain without red flag symptoms. Radiologia (Engl Ed). 2023. Vol. 65 Suppl 2: S59-S70. doi: 10.1016/j.rxeng.2023.02.004.

129. Bösch N., Hofstetter M., Bürki A., Vidondo B. Effect of Facetectomy on the Three-Dimensional Biomechanical Properties of the Fourth Canine Cervical Functional Spinal Unit: A Cadaveric Study Veterinary and Comparative Orthopaedics

and Traumatology. 2017. 30(6):430-437. DOI:10.3415/VCOT-17-03-0043

130. Pontoh L.A.P. et al. Combined exosome of adipose-derived mesenchymal stem cell and hyaluronic acid delays early osteoarthritis progression of ovine sheep model: Clinical, radiographic, macroscopic and microscopic evaluation. F1000Research 2024, 13:494. https://doi.org/10.12688/f1000research.147309.3

131. Moore S. A. Targeting Translational Successes through CANSORT-SCI: Using Pet Dogs To Identify Effective Treatments for Spinal Cord Injury. J Neurotrauma. 2017 Jun 15;34(12):2007–2018. doi: 10.1089/neu.2016.4745

132. Hinojosa R., Riggs L.C., Strauss M., Matz G.J. Temporal bone histopathology of cisplatin ototoxicity. Am J Otol. 1995 Nov;16(6):731-40.

133 Animal blood research and clinical interpretation of the obtained results: Methodological recommendations for students of the Faculty of Veterinary Medicine, managers and trainees of the Institute of Postgraduate Training of Managers and Specialists of Veterinary Medicine / V.I. Levchenko, V.M. Sokolyuk, V.M. Bezukh et al. – Bila Tserkva, 2002. 56 p.

134. Laboratory research methods in biology, animal husbandry and veterinary medicine: a reference book / V.V. Vlizlo, R.S. Fedoruk, I.B. Ratych and others; rev. V.V. Vlizlo, Lviv: SPOLOM, 764 p.

SCIENTIFIC EDITION

MONOGRAPH

NEUROLOGICAL SYNDROMES AFTER TRAUMA IN DOGS (ETIOLOGY, SYMPTOMS AND TREATMENT)

Authors:
R. Biloshytskyi, L. Kalachniuk, N. Grushanska, R. Bokotko, R. Dymko,
G. Tkach, L. Kladnytska, O. Shupyk, V. Rodz, P. Fedyshyn

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