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CLINICAL REVIEW

OSTEOARTHRITIS IN THE CAT
1. How common is it and how easy to recognise?

David Bennett, Siti Mariam bt Zainal Ariffin and Pamela Johnston

How prevalent is feline osteoarthritis?

The reported prevalence of feline osteoarthritis (OA) varies, potentially because different studies have involved different age groups of cats. The most important criterion for diagnosing radiographic OA is the presence of osteophytes, but radiographically normal joints can be affected by articular cartilage pathology; therefore, radiographic studies are likely to underestimate the prevalence of OA.

Hardie et al reported a 26% radiographic prevalence of appendicular OA and a 90% prevalence of all types of degenerative joint disease (DJD), but the study only included cats older than 12 years of age.[2] In a study from Utrecht University, the appendicular joints of 100 cats were radiographed, with no specific inclusion criteria other than the

Definitions

Osteoarthritis

OA is a common and complex progressive disease. Clinically it is defined as 'a slowly evolving articular disease characterised by the gradual development of joint pain, stiffness and the limitation of motion'. Pathologically it has been defined as 'an inherently non-inflammatory disorder of moveable (synovial) joints characterised by deterioration of articular cartilage and by the formation of new bone at the joint surfaces and margins'. It is often confusing and certainly misleading to refer to OA as a non-inflammatory disease but historically this has always been the case.[1]

Degenerative joint disease

DJD is often, incorrectly, used to mean the same as OA. DJD is an all-encompassing term that includes all types of degenerative pathology in any type of joint. For example, it includes OA but also spondylosis deformans of the intervertebral disc (non-synovial) joints, isolated degenerative lesions such as enthesiophytes, degenerative soft tissue mineralisation within joints which might not be part of OA (eg, of menisci, ligaments) and also traumatic arthritis.

Practical relevance Osteoarthritis (OA) is very common, particularly in older cats, but its clinical significance has largely gone unrecognised until recently. As in other species, OA is often painful and appropriate treatment is required to improve the animal's quality of life. Most cases appear to be primary or idiopathic. It is important for the clinician to actively seek these cases in the practice population.

Clinical challenges The recognition of chronic arthritic pain is a major challenge since most cats will not exhibit lameness. The main features of feline OA are changes in behaviour and lifestyle, which develop gradually and which owners tend to interpret as simply being the effects of old age. A meaningful physical orthopaedic examination can be difficult to achieve. A lack of familiarity with feline joint radiographs, and the fact that major cartilage pathology can be present in the absence of any bony change, mean that radiographic identification of OA in the cat can also be problematic.

Client questionnaire The recognition of chronic arthritic pain in the cat is based on owner questionnaires designed to elicit information about changes in mobility, activity levels, grooming habits and general demeanour.

Evidence base Several publications now report on the significance of behavioural and lifestyle changes as indicators of chronic arthritic pain in the cat. However, there is not as yet a fully validated owner-based questionnaire for recognising chronic pain in the cat. Furthermore, the aetiopathogenesis of feline OA still requires detailed investigation. Such studies are likely to make a major contribution to comparative rheumatology, since feline OA, more so than the canine disease, shows many similarities with human OA.

MANAGEMENT AND TREATMENT
Part 2 of this two-part article, reviewing pharmacological and other strategies for treating OA, appears on pages 76–84 of this issue of *J Feline Med Surg* and at:
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David Bennett
BSc BVetMed PhD DVM DSAO FHEA MRCVS*
Siti Mariam bt Zainal Ariffin
DVM
Pamela Johnston
BVM&S PhD PGCHE FHEA MRCVS
School of Veterinary Medicine,
University of Glasgow, Bearsden Road,
Bearsden, Glasgow G61 1QH, UK

*Corresponding author.
Email: David.Bennett@glasgow.ac.uk

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cats were aged greater than 6 years and all were cases referred to the university clinic, mainly for reasons unrelated to the musculoskeletal system:[3] 61% of the cats had OA in at least one joint and 48% had more than one joint affected. Of the cats that were over 14 years old, 82% had OA in at least one joint. Thus, the prevalence increased with age.

Investigators at Glasgow reported a prevalence of appendicular OA of 16.5% (and a prevalence of 33.9% for all types of DJD) in a general population of 218 cats aged from 0.2–18 years (mean age 6.5 years).[4] The median age of affected cats was 10.2 years, and increasing age was clearly a risk factor for the development of OA and other degenerative arthropathies. Of the 74 cats affected with DJD, 28.4% had both appendicular and axial DJD, 32.4% had only axial joints affected and 39.2% had only appendicular joints affected. The hip and elbow joints were most commonly affected and bilateral disease was invariably a feature. In over 60% of cases of feline OA, osteophytosis was categorised as mild, a feature also reported by Hardie et al,[2] which may suggest that osteophytosis is less evident in cats compared with other species. A similar retrospective study by Godfrey reported a 22% prevalence of radiographic OA (63 of 292 cats);[5] the affected population was again older than the control population. The elbow was the most commonly affected joint, similar to the Glasgow study, although many cats had more than one joint affected.

A more recent study by Lascelles et al of a randomly selected sample of 100 cats aged up to 20 years old found that almost all of the cats (92%) had radiographic evidence of DJD and that 91% had at least one site of appendicular DJD.[6] In contrast to previous studies, affected joints in descending order of frequency were hip, stifle, tarsus and elbow; however, some of the lesions that were included may not have fallen within the definition of DJD.

Is feline OA primary or secondary?

Most cases of feline OA appear to be primary or idiopathic; ie, there is no obvious underlying cause for the development of the disease.[1,2,4–6] Secondary OA is where the disease is associated with some other existing joint pathology.

Secondary OA following joint trauma and hip dysplasia is well recognised.[2–4] Clarke et al suggested that approximately 25% of OA cases resulted from trauma,[4] and Godfrey reported approximately 13% were trauma related.[5] However, it is very difficult to confirm or rule out the occurrence of joint trauma during the lifetime of an individual animal. Many cases of joint trauma may go unnoticed by owners and it may be repetitive low-grade trauma over a period of time that is the important factor.

Other underlying causes of secondary feline OA include mucopolysaccharidosis,[7] medial patellar luxation,[8] acromegaly,[4,9] Scottish fold osteochondrodysplasia,[10] developmental luxation of the radial head,[11] and other arthropathies.[12] Cranial cruciate ligament failure in the cat may also occur as a gradual 'non-traumatic' event and lead to secondary OA.[13] Certain breeds are prone to hip dysplasia (eg, the Maine Coon)[14,15] and therefore a high prevalence of hip OA would be expected. The Burmese breed appears predisposed to elbow OA.[16] Hypervitaminosis A is a specific form of DJD associated with the feeding of liver-rich diets, which can affect synovial joints, but the pathology differs from that of OA.[17] Use of the term 'primary' or 'idiopathic' OA may just hide an ignorance of the underlying aetiology. There may also be, as yet unidentified, genetic abnormalities that predispose an individual animal to premature degeneration of a joint.

The main risk factor for both increasing prevalence and severity of feline OA is advancing age. It is uncertain what role obesity plays in the pathogenesis of feline OA.

What are the risk factors?

The main risk factor for both increasing prevalence and severity of feline OA is advancing age.[3–6,18] Obesity has been shown to be a risk factor in other species but at present this has not been confirmed in the cat.[3,4,6,18] Only approximately 14% of older cats with painful OA are obese,[18] although the relevance of obesity in younger cats where OA is not a pathological and/or clinical problem is not known.[19–22] Obesity in the older cat undoubtedly worsens the clinical problem by resulting in 'mechanical overload' of the diseased joint. However, obesity is increasingly becoming implicated more directly in the pathogenesis of human OA by contributing to synovial inflammation and chondrocyte damage.[23–25] Adipose tissue is now regarded as an important secretory and endocrine gland that secretes many cytokines associated with cartilage degeneration (TNF-alpha, IL-1, IL-6), as well as fat-specific hormones such as leptin and adiponectin which can promote cartilage degeneration.[23,26]

Lascelles et al reported several possible associations with DJD, although once age had been accounted for there were no variables that were significantly associated with DJD.[6] However, there remains the possibility that these associations might indeed exist and be important in the pathogenesis. For example, these authors reported a strong association between DJD and blood cholesterol levels, and this might reflect the influence of lipid metabolism in the development of OA.

What are the clinical features?

Many authors have reported a mismatch between radiographic and clinical signs. Although 90% of the cats in the study by Hardie et al had radiographic DJD, only 4% were lame.[2] Godfrey reported that of 63 cats with radiographic appendicular OA, only 11 had a limp, and another five exhibited stiffness.[5] Clarke et al reported that only 16.7% of cats with radiographic OA were lame, and suggested that lameness may not be a major clinical sign of painful arthritic disease in the cat.[4] This is supported by the findings of an experimental study in the cat involving transection of the cranial cruciate ligament, which found that lameness was not the major feature, even after 1 year when significant radiographic OA had developed.[27]

A prospective study of 28 cases of feline OA by Clarke and Bennett found that most cats showed alterations in their behaviour and lifestyle when affected by OA, which improved significantly after analgesia.[18] The most significant lifestyle changes were a reduced ability to jump (20/28 cats) and reduced activity levels (17/28). In a subsequent study, an owner questionnaire was used to gather information on mobility, activity, grooming and temperament, before and after the cat was administered a 28-day course of meloxicam (see box below).[28] Owners were asked to score their cat's current lifestyle/behaviour compared with how it was when it was a young adult, and then to make the same comparison after the course of treatment. Owners reported a change in their cat's

Owner behaviour watch[28]

Think about how your cat used to be and compare this with how he/she is now, using the type of activities listed below as a guide and grade the severity of change 1–10.

After starting treatment, think about any changes that have occurred since the start of treatment, but again grade severity of any behavioural change by comparing with when he/she was a young adult.

Table 1 Examples of behaviours to consider for evaluation when trying to identify cats suffering chronic arthritic pain

	Behaviour	Change
Mobility	Jumping up or down	Refusing or hesitating to jump UP or DOWN Less agile on stairs No longer attempting to reach high spots
	Size/height of jump, up or down	Makes smaller jumps (eg, takes several steps to reach high spots) Frequency of jumps (eg, jumps onto high surfaces less than before)
	Gracefulness	Movement less graceful than before Becoming stiff and 'creaky'
	Changes in toileting	Changes in location (eg, reluctant/refusing to go outside or reluctant/refusing to use litter tray) Difficulties using litter tray (eg, missing tray sometimes/often)
Activity levels	Sleeping habits	Sleeping or resting more Lying in the same spot for a long time, not moving location often Changes in resting place
	Playing	Playing less No longer instigating play More difficult to tempt to play
	Hunting	Hunting less than before
Grooming habits	Coat condition	Coat matted or scurfy, generally or in one particular place Observed grooming behaviour less frequent or of shorter duration 'Over-grooming' in certain areas
	Scratching	Sharpens claws less frequently Change of location/height where scratching occurs Claws overgrown or catching on carpets or clicking on hard floors
Temperament (demeanour)	Tolerance to owner or other animals	Less keen to interact Grumpy on contact with other cats Grumpy on contact with other animals including owner
	General attitude	Quieter Spending more time alone Not seeking/avoiding contact with other cats or other animals Not seeking/avoiding contact with owner

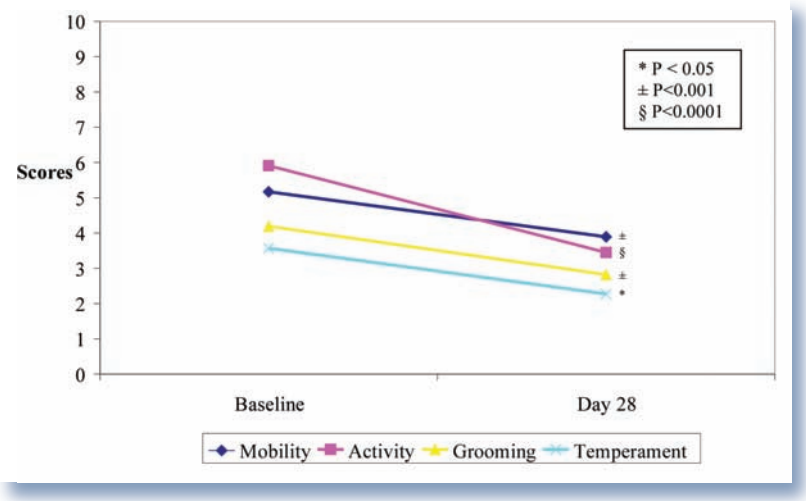


Figure 1 Change in mean scores for each of four behavioural domains in cats with OA between baseline and following 28 days of treatment with an analgesic (meloxicam) (Bennett and Morton).[28] The higher the score, the more change there has been in the parameters listed in Table 1 as compared with the situation when the cat was a young adult; the lower score after 4 weeks of analgesic therapy means less change and thus the cat is behaving more like its old self. Owners can easily identify these changes and assess/score the degree of alteration

behaviour and lifestyle over time, and a reverse in these behavioural patterns when pain relief was given (Figure 1); the greatest changes were noted in the activity and mobility domains. Changes in jumping behaviour are particularly obvious to owners.

A similar protocol, including a placebo group, was used by Lascelles et al in a study of 13 indoor cats that were older than 10 years of age.[29] These authors used 'client-specific outcome measures' as a subjective assessment, in combination with collar-mounted accelerometers as an objective measurement. Owners reported a significant improvement in their cat's activity levels and quality of life after meloxicam compared with placebo, although there was also some improvement in activity with placebo. The accelerometer data showed a significant increase in activity levels after meloxicam, but not placebo. The same group subsequently reported a further study where 100 randomly selected cats aged between 6 months and 20 years old were assessed for radiographic DJD and pain, and two 'extreme' groups were identified by veterinary assessment, one with severe disease and one with mild disease.[30] The owners of both groups were asked to rate the ability of their cats to perform various activities. Significant differences in the owners' answers were identified for 17 out of 28 questions, with changes in activity and mobility levels being the most apparent.

Slingerland et al studied 100 cats aged 6 years or older, and used a questionnaire to identify behavioural and lifestyle changes associated with OA.[3] They reported decreased mobility (including less jumping, decreased height of jump, stiffness and problems walking up and down stairs) and less grooming as being associated with OA, although these features also both strongly correlated with increasing age.

Joint thickening, synovial effusion, reduced range of motion and crepitus are far less obvious in the arthritic cat compared with the dog.

Increased elimination directly over the edge of the litter tray was also associated with OA and did not correlate with increasing age.

A thorough physical examination is helpful, but often difficult

A careful physical examination is important, but challenging.[1] The examination must take place in a quiet, secure and stress-free environment and the use of pheromone sprays or diffusers in the waiting and consulting rooms can help to relax the cat. It is useful to try and assess the cat's gait in the consulting room, but this is difficult. Cats do not walk in straight lines, are not usually trained to the lead and are generally more interested in investigating the unfamiliar environment or seeking somewhere to hide. In some cases it is possible to assess their willingness to jump. Palpation and manipulation of the joints must be done carefully and it is not unusual for some cats to resent these procedures even if joints are normal and pain free.

Joint thickening, synovial effusion, reduced range of motion and crepitus are far less obvious in the cat compared with the dog. Pain is most often elicited on extremes of joint motion, but is very difficult to assess in this species. If a cat becomes aggressive and uncooperative during the examination there is no benefit in continuing; a second attempt can be made later. The examination should include an assessment of spinal pain, particularly lumbar and lumbosacral pain, since cats with spondylosis can show behavioural changes similar to those seen with OA.

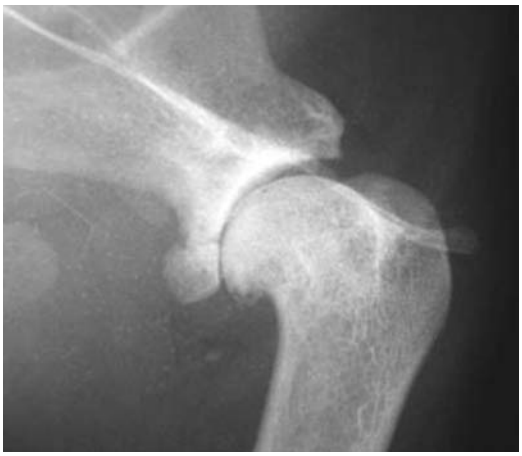
What are the radiographic features?

The radiographic features of OA and DJD in the cat have not been extensively described, although the study by Lascelles et al has provided useful information.[6] Because the importance of feline OA was not recognised until very recently, most clinicians lack the experience of regularly looking at articular radiographs of cats and recognising abnormalites. As in the dog, the presence of osteophytes is regarded as the key radiographic feature of OA, although these can be difficult to identify or even absent. It is not unusual to see ossicles and soft tissue mineralisation within feline arthritic joints. Soft tissue thickening and synovial effusion can be evaluated by radiography but are less apparent in feline OA than in other species. Radiographically normal joints can be pathologically and clinically affected, and radiographically affected joints can be pain free.

Shoulder joint

OA of the shoulder joint is most easily diagnosed by identifying osteophyte formation on the caudal rim of the glenoid and on the caudal edge of the humeral head (Figure 2). Osteophytes are always more readily seen when they protrude from the surface of the bone. However, osteophytes tend to develop around the whole joint structure and thus they may also appear as a sclerotic line along the glenoid and humeral head, generally corresponding to where the joint capsule attaches. It is not unusual to see an apparent line of separation between the osteophyte and the edge of the glenoid (Figure 2), and this may represent a situation where the osteophyte has not become completely incorporated within the epiphysis, or it may represent, rather than an osteophyte, an osteochondroma (separate mineralised body) (Figure 3a,b).

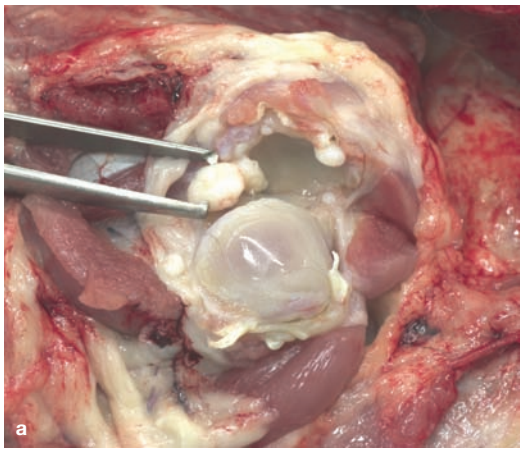
Figure 2 Mediolateral radiograph of the left shoulder of a cat with OA. There is sclerosis of the glenoid and the glenohumeral joint space appears reduced at its caudal aspect. Osteophytes are seen on the caudal aspect of the humerus. The apparent separate mineralised body at the caudal edge of the glenoid could be an osteophyte not completely remodelled or could represent a 'free' osseocartilaginous body (osteochondroma), as seen in Figure 3. Note the clavicle



The presence of osteophytes is regarded as the key radiographic feature of OA, although these can be difficult to identify or even absent.

Osteochondromas are regularly seen within arthritic joints of cats on pathological examination, particularly within the elbow and shoulder, but mostly they are not sufficiently mineralised to be visible on radiography. Osteochondromas can be completely free within the articular cavity, free but embedded within the surface of the synovium, or actually attached to the synovium (Figure 3a).

It is sometimes possible to appreciate a reduced joint space on the caudal aspect of the joint due to cartilage loss, but assessing joint space is always a problem in small animals where non-weightbearing views are used. The clavicle is a very obvious feature of the feline shoulder joint and should not be confused with pathological mineralisation or osteophyte development. The cat also has a very prominent coracoid process of the glenoid and its appearance is influenced by radiographic positioning; it is easily mistaken for new bone formation on a caudocranial or craniocaudal film.



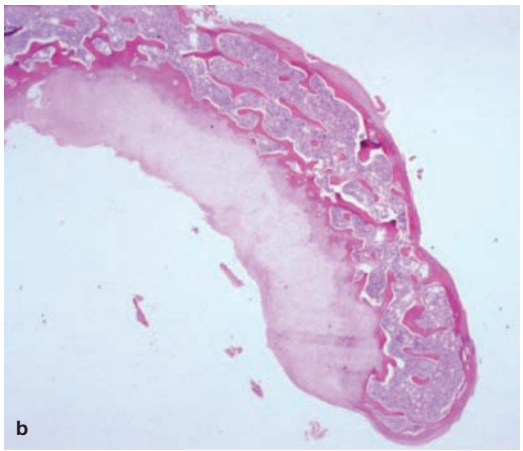


Figure 3 **(a) Photograph of a feline shoulder showing advanced OA. There is an extensive area of total cartilage loss on the humeral head with eburnation of the underlying subchondral bone. Several discrete osseocartilaginous bodies (osteochondromas) are seen on the surface of the synovium; some of these are totally free and mobile; others are 'adherent' to the synovium. The forceps have lifted an 'ossicle' from a fossa within the synovial surface in which it was embedded. (b) Photomicrograph of an osteochondroma originally embedded within the synovial membrane of the elbow joint. Note the presence of trabecular bone despite no obvious blood supply being present, and the cartilaginous ('articular') surface which contacted the distal humerus**

Elbow joint

Radiographic and associated pathological features of feline elbow OA are illustrated in Figures 4–8.

Osteophyte formation is again a key feature and this may be seen at various sites within the elbow joint: radial head, medial coronoid process, and distal humerus (particularly caudally and medially). Although subjective, the presence of subchondral bone sclerosis beneath the ulnar trochlear notch is another key feature of elbow OA (Figures 4, 6 and 8). Sclerosis is more easily assessed in the feline patient compared with the dog since there is less variation in anatomy between different types and breeds of cat, and thus it is easier for the clinician/radiologist to appreciate changes in bone opacity. Apparent increased radiopacity of bone can be due to increased thickening of the bony trabeculae beneath the articular surface, to osteophyte formation along the articular margin, or to soft tissue mineralisation within the joint capsule and/or intra-articular mineralised bodies (osteochondromas) superimposed on the epiphyseal bone (Figures 4–8).

Radiographically normal joints can be pathologically and clinically affected, and radiographically affected joints can be pain free.

Soft tissue mineralisation is common in elbow OA, particularly in advanced cases (Figures 4 and 8). Occasionally, an extensive bony reaction on the medial epicondyle may be seen. This has been referred to as 'medial epicondylitis' and appears to be associated with the attachment of the flexor muscles,[31] but is generally part of a more extensive elbow OA. It is important to note the existence of a sesamoid bone within the supinator muscle of the feline elbow joint, which is generally present in all elbows but is radiographically visible in only about 40% of normal joints.[32] This sesamoid bone does become more obvious (mineralised) in an arthritic joint and is often increased in size (Figures 4 and 8). Occasionally, there appears to be two or more sesamoid bones in the arthritic elbow, although it can be difficult to distinguish a sesamoid bone from soft tissue mineralisation (Figure 8).

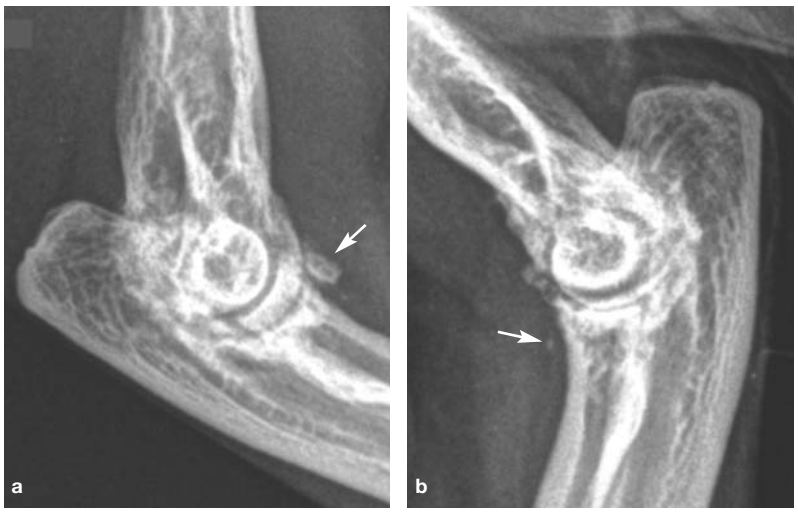


Figure 4 Mediolateral radiographs of the left (a) and right (b) elbow. There is osteophyte formation on the distal humerus of the right elbow. Apparent sclerosis beneath the ulnar notch is present in both elbows. The supinator sesamoid bone is easily seen in the left elbow (arrow) but is less apparent in the right. The arrow on the right elbow shows soft tissue calcification within the joint capsule

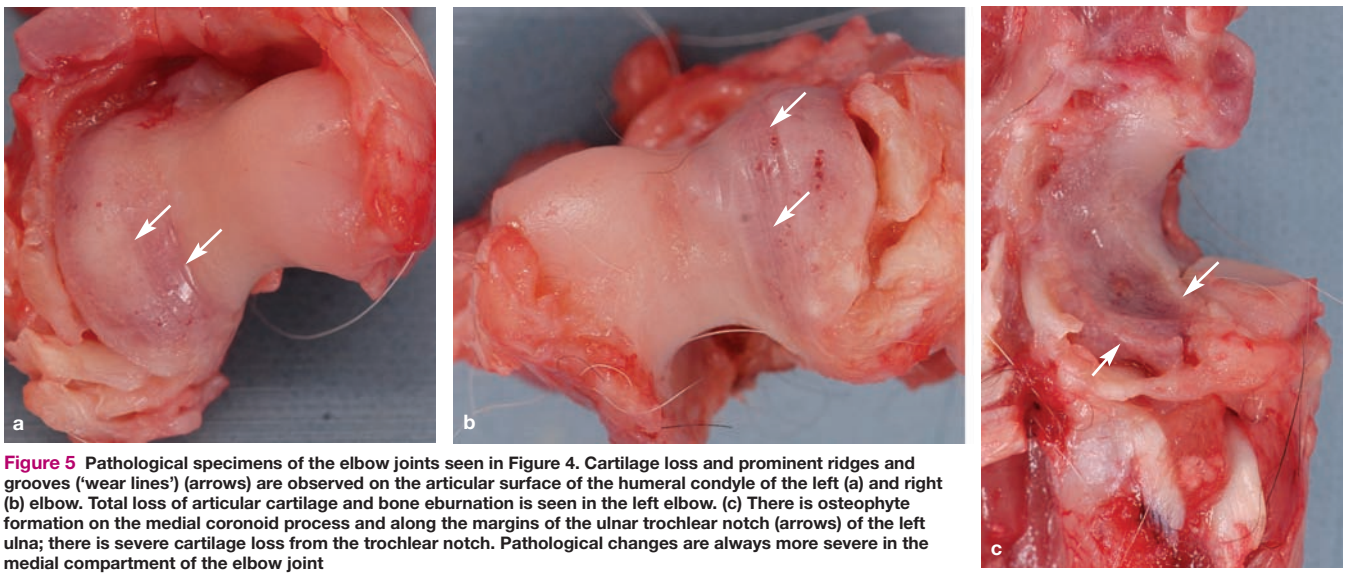


Figure 5 Pathological specimens of the elbow joints seen in Figure 4. Cartilage loss and prominent ridges and grooves ('wear lines') (arrows) are observed on the articular surface of the humeral condyle of the left (a) and right (b) elbow. Total loss of articular cartilage and bone eburnation is seen in the left elbow. (c) There is osteophyte formation on the medial coronoid process and along the margins of the ulnar trochlear notch (arrows) of the left ulna; there is severe cartilage loss from the trochlear notch. Pathological changes are always more severe in the medial compartment of the elbow joint

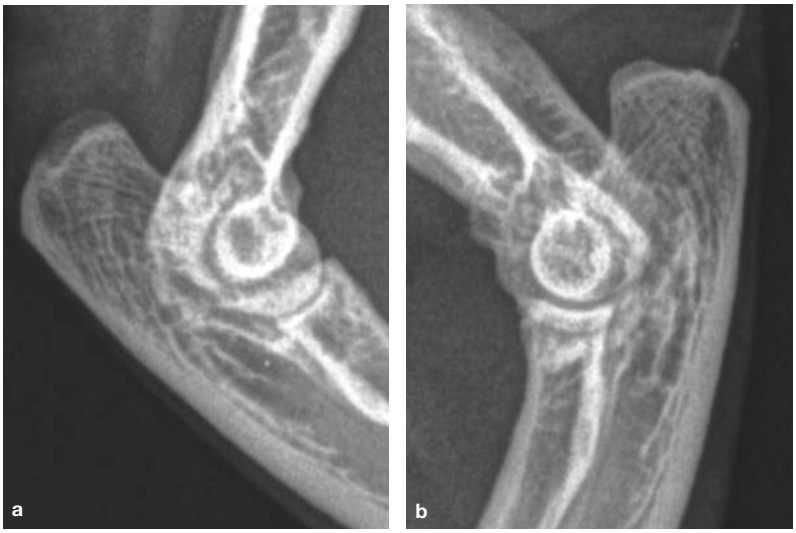


Figure 6 Mediolateral radiographs of the left (a) and right (b) elbow joint. There is a mild degree of increased radiopacity beneath the ulnar trochlear notch, particularly in the right

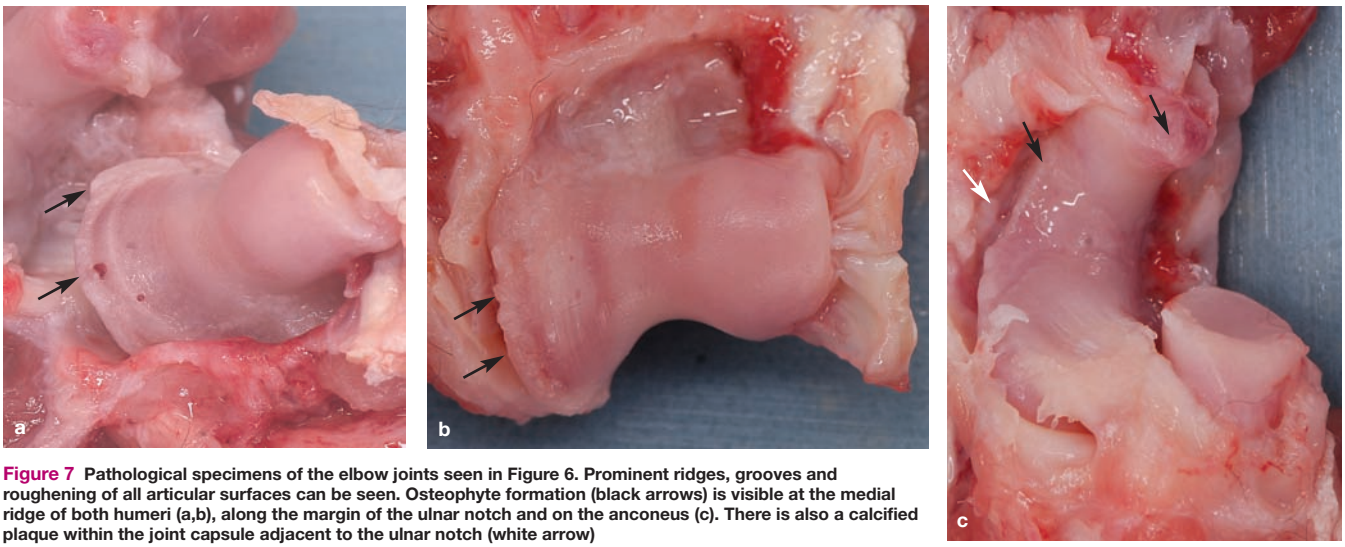


Figure 7 Pathological specimens of the elbow joints seen in Figure 6. Prominent ridges, grooves and roughening of all articular surfaces can be seen. Osteophyte formation (black arrows) is visible at the medial ridge of both humeri (a,b), along the margin of the ulnar notch and on the anconeus (c). There is also a calcified plaque within the joint capsule adjacent to the ulnar notch (white arrow)

The most severe pathology appears to occur within the medial compartment of the joint (Figures 5, 7 and 8).

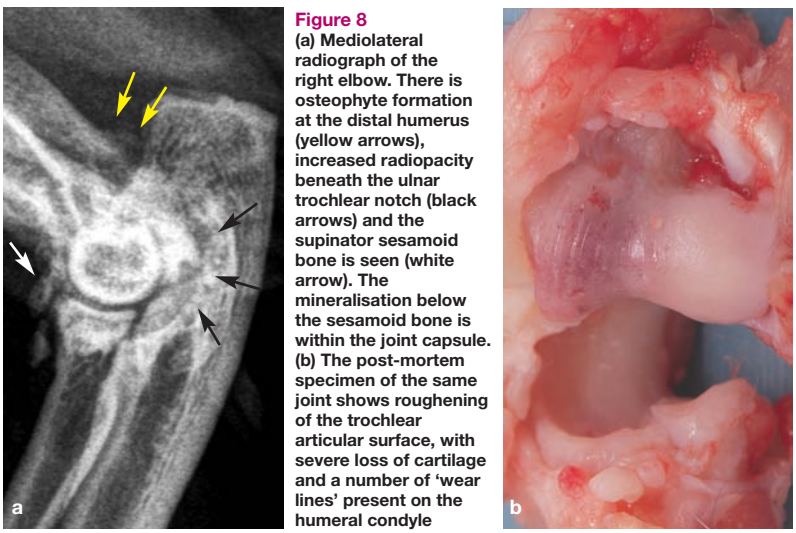


Figure 8 (a) Mediolateral radiograph of the right elbow. There is osteophyte formation at the distal humerus (yellow arrows), increased radiopacity beneath the ulnar trochlear notch (black arrows) and the supinator sesamoid bone is seen (white arrow). The mineralisation below the sesamoid bone is within the joint capsule. (b) The post-mortem specimen of the same joint shows roughening of the trochlear articular surface, with severe loss of cartilage and a number of 'wear lines' present on the humeral condyle

Hip joint

Although hip dysplasia is well documented in the cat it only accounts for about 20% of cases of hip OA.[18] This, of course, is influenced by how hip dysplasia is defined; at present the authors define hip dysplasia as less than 50% coverage of the femoral head by the dorsal acetabular edge on the extended ventrodorsal view. Joint laxity has been demonstrated in feline hip dysplasia,[33] as it has in the dog, but more research is required into the relationship between feline hip laxity, hip dysplasia and arthritis. It is likely that if joint laxity is used to diagnose feline hip dysplasia, the prevalence will be much increased.

Keller et al reported a 6.6% prevalence of hip dysplasia in a hospital population of cats and demonstrated no statistical difference in the prevalence of hip dysplasia between domestic shorthair and purebred cats and no effect of sexual status.[14] However, the Maine Coon breed is reported as having a much

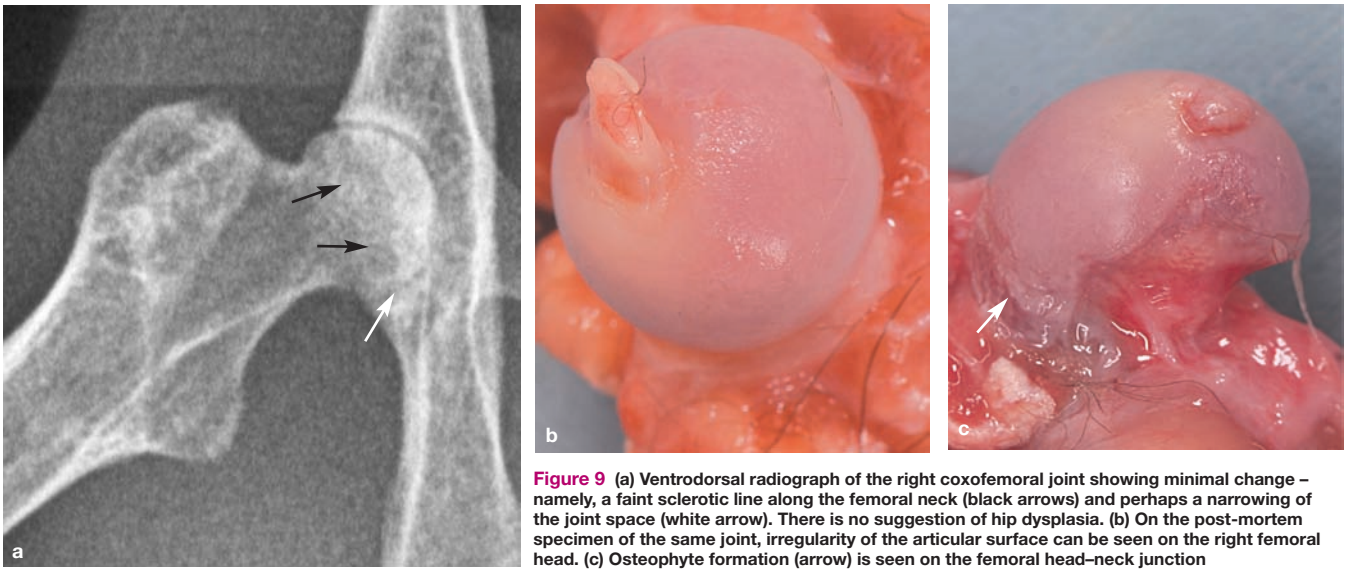


Figure 9 (a) Ventrodorsal radiograph of the right coxofemoral joint showing minimal change – namely, a faint sclerotic line along the femoral neck (black arrows) and perhaps a narrowing of the joint space (white arrow). There is no suggestion of hip dysplasia. (b) On the post-mortem specimen of the same joint, irregularity of the articular surface can be seen on the right femoral head. (c) Osteophyte formation (arrow) is seen on the femoral head–neck junction

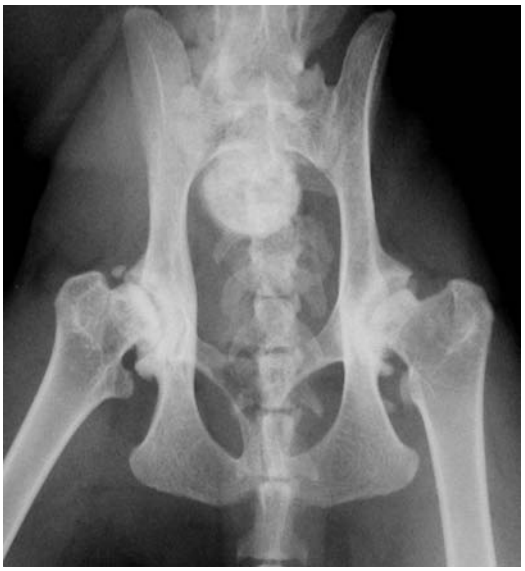


Figure 10 Ventrodorsal radiograph of the pelvis showing extensive remodelling of both hip joints. Osteophyte formation is seen on the cranial and caudal acetabular edges and there is sclerosis affecting both femoral necks, consistent with osteophyte formation. Both femoral heads are misshapen, which may represent remodelling or a particular type of dysplasia. In this case hip dysplasia is present

higher prevalence, and genetic influences are certainly thought to be involved in feline hip dysplasia.[34] Clarke and Bennett recorded a Norberg angle for the dysplastic hip of 87.5°, compared with 99.2° for the normal joint.[18] The normal Norberg angle is much less in the cat than the dog, indicating a shallower acetabulum.

OA of the hip joint is characterised by osteophyte formation, particularly on the cranial effective acetabular rim and the femoral neck, where it often appears as a sclerotic line (Figures 9 and 10). Loss of joint space is another feature (Figure 9).

Stifle joint

OA of the stifle joint is characterised by osteophyte formation on the patella along the trochlear margin (where again it appears as a sclerotic line) and on the caudal edge of the tibia (Figure 11). Soft tissue mineralisation is

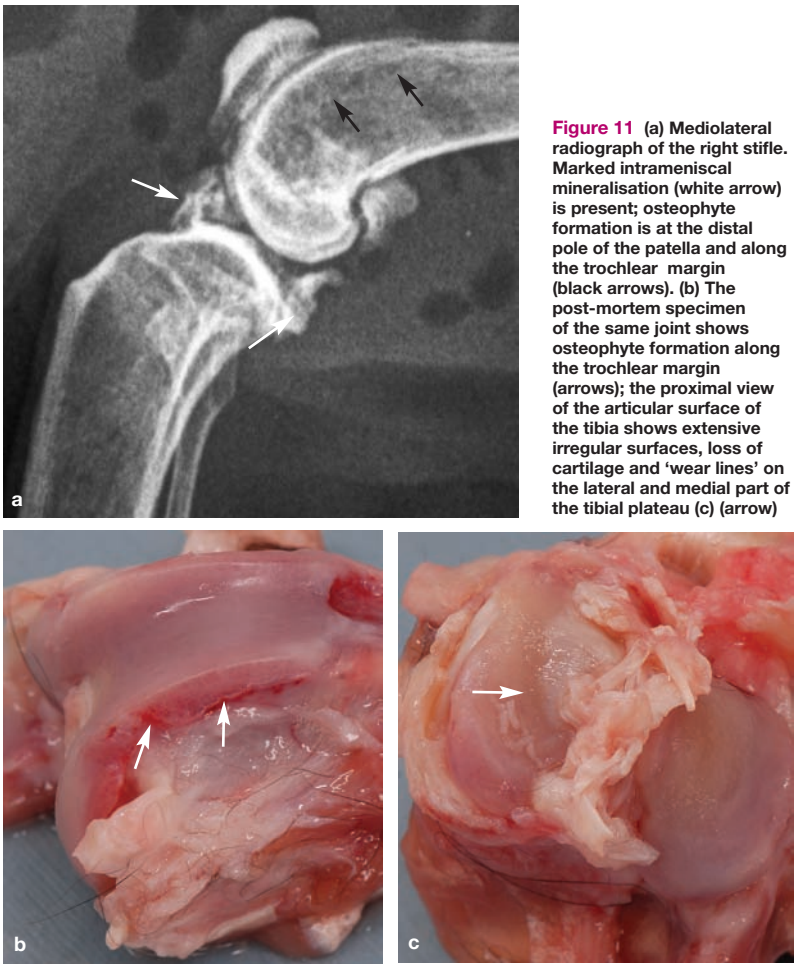
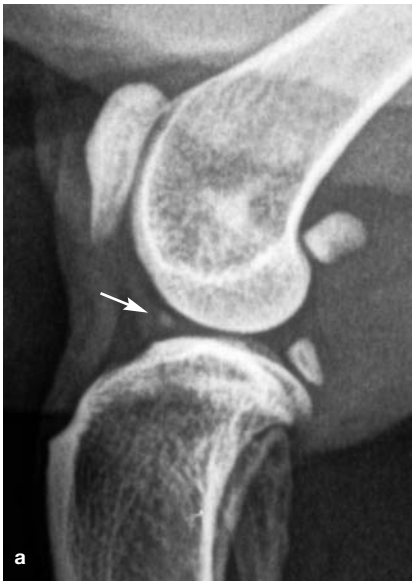


Figure 11 (a) Mediolateral radiograph of the right stifle. Marked intrameniscal mineralisation (white arrow) is present; osteophyte formation is at the distal pole of the patella and along the trochlear margin (black arrows). (b) The post-mortem specimen of the same joint shows osteophyte formation along the trochlear margin (arrows); the proximal view of the articular surface of the tibia shows extensive irregular surfaces, loss of cartilage and 'wear lines' on the lateral and medial part of the tibial plateau (c) (arrow)



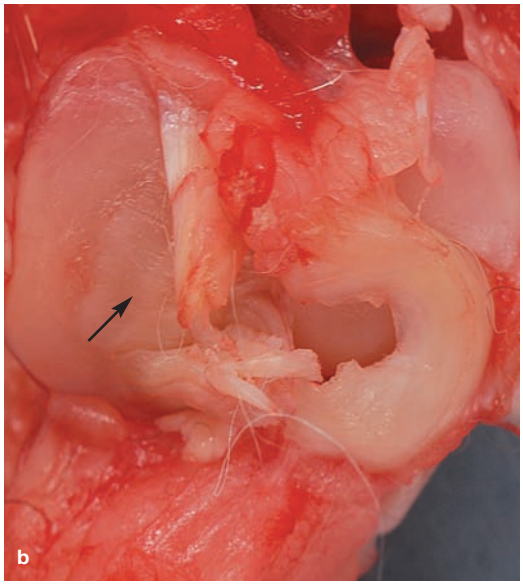


Figure 12 (a) Mediolateral radiograph of the right stifle joint. Intrameniscal mineralisation (arrow) is seen. Note only one fabella is visible, which is not unusual in the cat. (b) The post-mortem photograph of the same stifle joint shows an irregular articular surface with a number of 'wear lines' running transversely on the medial part of the tibial plateau (arrow)

also often seen. In particular it is common to see mineralisation within the articular space on the mediolateral view (Figures 11–13). This mineralisation appears to be within the cranial pole of the medial meniscus and it may represent a degenerative calcification within the meniscus as part of the arthritic process.

However, in some cases this mineralisation is thought to represent a meniscal sesamoid bone, the lunula.[35] In a study by Freire et al, 46% of client-owned cats had radiographically apparent meniscal mineralisation, although there was no difference in pain scores between those joints with mineralisation and those without.[36] This study also looked at 30 cadavers and 34 of 57 stifle joints had mineralisation of the medial meniscus. The age of these cats was not recorded but the presence of mineralisation was associated with articular cartilage degeneration within the medial compartment of the joint. This intrameniscal mineralisation can certainly be seen in very young cats and where there is no other evidence of OA. The authors believe that the meniscal mineralisation does increase with age and with arthritic change, but further studies are required to decide whether these mineralisations are always pathological or whether they might represent normal anatomical structures that could become a focus of additional calcification when the joint becomes arthritic.

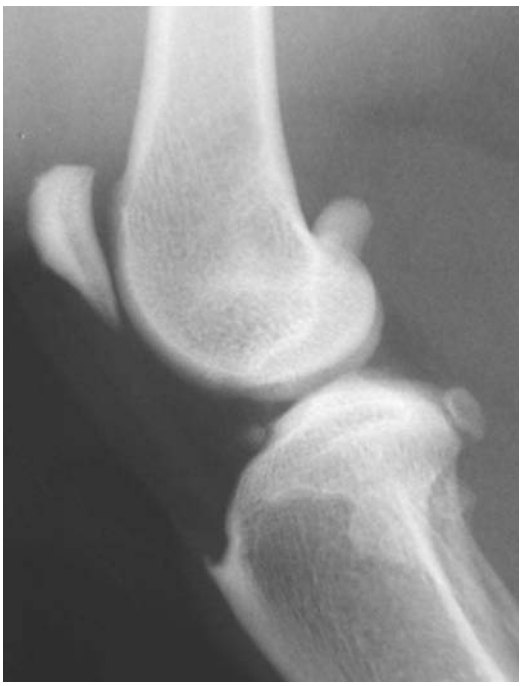
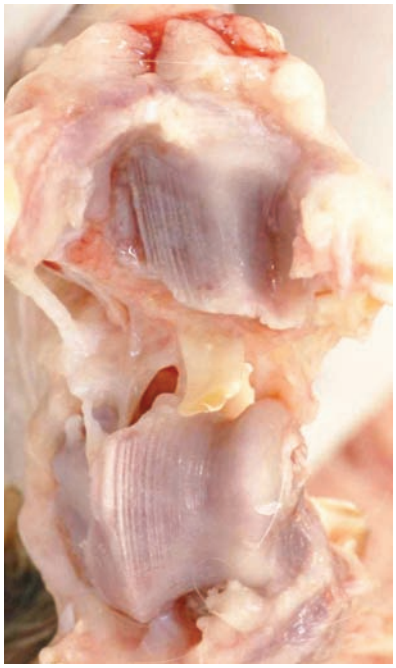


Figure 13 Mediolateral radiograph of the stifle joint. Intrameniscal mineralisation is present. There is also a spur of bone where the patellar ligament attaches to the tibial tuberosity (enthesiophyte). No osteophyte formation can be seen. The tibial enthesiophyte is not uncommonly seen in the feline stifle and, although it can be part of OA, it probably represents an isolated traumatic lesion in this case and is of doubtful clinical significance. Again, only one fabella is visible

Mineralisation within the cranial cruciate ligament has also been documented, as has enthesiophyte formation at the attachment of the patellar ligament on the tibial tuberosity (Figure 13). The latter may also be part of stifle OA but may occur as a solitary lesion (presumably traumatic in origin) which is of no or little clinical significance.

It is rare to see both fabellae in the cat's stifle; generally only the lateral one is visible (Figures 12 and 13). The medial fabella is present but is often not sufficiently mineralised to show on a routine radiograph. Again, the medial fabella is possibly more likely to be seen in an arthritic joint where new bone deposition may occur as a result of the arthritic process.

Pathological changes are most evident within the medial compartment of the femorotibial joint (Figures 11 and 12), but the patellofemoral joint may also be affected.



Hock and carpus

OA of the hock is characterised mainly by osteophyte formation on the distal tibia and on many of the tarsal bones, together with soft tissue mineralisation. Most of the pathology in the feline hock joint appears to occur within the lateral compartment (Figure 14). OA of the carpus is rare in the cat, although osteophyte formation may occasionally be seen on the distal radius and on the carpal bones.

Figure 14 **Post-mortem specimen of the tibiotarsal joint. There is loss of articular cartilage and several 'wear lines' in the articular surfaces of both the distal tibia and tibial tarsal bone. Most pathology is affecting the lateral compartment of the joint**

KEY POINTS

- It is a poor reflection on the veterinary profession that OA of the cat has been unrecognised for so long.
- It is a common disease but seldom produces overt lameness. It is not entirely clear why lameness is a rare sign but it could be related to the bilateral nature of feline OA, the agility of the species, making it more able to cope with its articular pain, or the fact that the domestic cat, like its free-living ancestors, will try to mask any weakness such as lameness to protect itself from predators.
- Changes in the cat's lifestyle and behaviour are important indicators of a potential problem, particularly reduced activity levels and unwillingness to jump.
- The owner is of paramount importance in alerting the veterinarian to the possibility of chronic arthritic pain in their pet. However, most owners believe that their cat's behavioural changes are simply related to increasing age ('slowing down'), are to be expected, and do not indicate that their pet is in pain.
- It is recommended that owners of all cats over 6 years of age are asked to complete a questionnaire (such as that shown on page 67) as part of a routine 'health check' once or twice a year. This is best done with the veterinarian or practice nurse and the practice should be proactive in contacting relevant clientele.
- All older cats coming to the surgery, whatever the reason, should also be included in this questionnaire-based health check.

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Conflict of interest

The authors declare that there is no conflict of interest.

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