

Case Log File – 200 CLINICAL CASES – PLEASE SEND IT AS PDF VERSION AND LANDSCAPE

Resident's Name:			
Resident's Signature		Date:	
Supervisor			
	I certify that I have reviewed this resident's case log and I confirm that the case log fulfils the guidelines as requested		
Supervisor Signature:		Date:	
Host Institution			
Residency Start Date:			
Resident End Date:			

Case log needs to be submitted as a PDF file. Word files will be returned to the candidate without assessment.

Guidance for Completion of the Case Log File (200 CLINICAL CASES):

Signalment key: Male: M, Female: F, Entire: E, Neutered: N. Age in years and months.

Neuroanatomical localisation: classify the neuroanatomical localisation into one of the following regions and then provide further details: Brain - Forebrain (Cerebral cortex, Thalamus), Brain - Brainstem (midbrain, pons, medulla oblongata), Brain - Cerebellum, Spinal Cord (C1-C5 spinal cord segments (SCS), C6-T2 SCS, T3-L3 SCS, L4-S3 SCS), Hyperaesthesia without neurological deficits, Cranial nerves, Neuromuscular (Peripheral neuropathy, neuromuscular junction, myopathy), Multifocal. Candidates must ensure that the cases included in their case log **are distributed evenly** over these neuroanatomical regions.

Investigation and diagnosis: describe the investigation under the following subheadings (where appropriate to the case): Radiography; Myelography; CT; MRI; CSF; Electrodiag (recommended case number:10, minimum expected case: 5, effective for residencies starting 1st January 2027); Clinical path; Histopath; Other. Ideally, all the investigative categories listed above are represented in the case log and this will be reflected in the assessment. The candidate must outline with a depth of coverage the results of the investigations and diagnosis with no identifiable errors or omissions. The description of the imaging findings must be well described, synthesize and outline with the key points mentioned. **The cases log should have significant number of cases with histopathological diagnosis.**

Treatment: candidates must indicate whether the cases were managed medically or surgically. In surgically managed cases, candidates must indicate whether they were the primary surgeon, assistant surgeon, observing the surgery or not involved in the surgery. The key points of the treatment should be well described, synthesized and outlined.

Outcome: Candidates must ensure that **all clinical cases have a minimum of follow up**. Cases with lost to follow up should be kept to none or to the minimum.

All cases must have been seen during the residency training period (months and years in agreement with the residency programme accepted by the Education Committee)

Clinical cases must be correctly summarised with the essential points. **The Case log must be presented as 2 to 4 clinical cases per page.**

Please **submit** the case log with **this format and converted to PDF**.

1. ABBREVIATIONS (Create a list of abbreviations in order to abbreviate your case log as much as possible)

[illegible]

2. CONTENTS

Small Animals

Brain - Forebrain (Cerebral cortex, Thalamus)

N° of cases: __ (__ %) __

Brain - Brainstem (midbrain, pons, medulla oblongata)

N° of cases: __ (__ %) __

Brain – Cerebellum

N° of cases: __ (__ %) __

Spinal Cord

N° of cases: __ (__ %) __

C1-C5 spinal cord segments

Cases should be distributed evenly between forebrain, multifocal, brainstem, spinal cord segments and neuromuscular system. Brainstem and neuromuscular cases may have less case numbers but still expected to be represented in substantial numbers

N° of cases: __ (__ %) __

C6-T2 spinal cord segments

N° of cases: __ (__ %) __

T3-L3 spinal cord segments

N° of cases: __ (__ %) __

L4-S3 spinal cord segments

N° of cases: __ (__ %) __

Cerebellar, Diffuse CNS, hyperaesthesia without neurological signs and cranial nerves groups does not need to be equally distributed with the above group.

Diffuse CNS (for instance: tetanus)

N° of cases: __ (__ %) __

Hyperaesthesia without neurological deficits

N° of cases: __ (__ %) __

Cranial nerves

N° of cases: __ (__ %) __

Neuromuscular (Peripheral neuropathy, neuromuscular junction, myopathy)

N° of cases: __ (__ %) __

Multifocal

N° of cases: __ (__ %) __

Other Animals (excluding canines and felines) **10% or 5% if performed rotation**

N° of cases: __ (__ %) __

Neurosurgeries (**Resident does not have to be the primary surgeon**) **25%**

N° of cases: __ (__ %) __

Electrophysiology (Recommended 10 cases, minimum 5 cases)

N° of cases: __ (__ %) __

Please read the file “Guidelines – Marking Grid Case Log” ([link](#)) to familiarise yourself on what is being assessed.

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#	Exam Date (dd/mm/yy) ID and Signalment	Neurological Examination	Neuroanatomic Localisation	Investigation	Diagnosis	Treatment	Outcome
1	22/02/15, 123123, Canine, French bulldog, MN/5y2m	History: 2 w progressive R sided head tilt, vestibular ataxia, R facial paralysis and R sided chronic ear disease Neurological examination: R sided head tilt, mild vestibular ataxia to the R, R facial nerve paralysis with neurogenic KCS, mild postural deficits in R TL and R PL.	Brain – Right medulla oblongata (right central vestibular syndrome and right facial neuropathy)	Clinical pathology: Biochemistry and CBC unremarkable, MRI: T2W hyperintense and T1W contrast enhancing material in the right middle ear with focal, ill defined T2W and FLAIR hyperintense, mildly contrast enhancing lesion in the medulla oblongata. Osteolysis of the right bulla wall with extension of the inflammatory process into the adjacent soft tissues. Reactive right medial retropharyngeal lymph node CSF (cisternal): TNCC 68 mm³ (n<6), RBC 11 mm³ (n<0), Protein(micro) 0.49 g/l (n<0.3), neutrophilic pleocytosis. No evidence of microorganisms. Microbiology (intraoperative): Scant growth of <i>Staphylococcus pseudintermedius</i> after 48h. Sensitive to enrofloxacin.	Right ascending bacterial otitis media and interna with focal meningoencephalitis	Surgical: Right Total Ear Canal Ablation and Lateral bulla osteotomy was performed (not present for Sx). Enrofloxacin for 21 d.	Time of discharge (4 d PSx): stable 2 w PSx improved, R sided facial nerve paresis; R sided vestibular syndrome with no evidence of central involvement 2 y PSx Extensive treatment of various skin issues by the dermatology team over last 2 y, mild R sided head tilt remained
2	13/04/15, 124561, Canine, Cross breed - medium, MN/10y5m	History: peracute onset non progressive L lateralizing paraparesis after jumping over a fence. Neurological examination: L lateralizing ambulatory paraparesis with proprioceptive ataxia, increased tone in R PL and decreased tone in the L PL, mild delay in paw positioning and hopping in R PL, absent in L PL, decreased withdrawal reflex L PL, L lateralizing cutaneous trunci reflex cut off at L2 vertebra, no spinal hyperaesthesia	Spinal Cord , T3 - L3 SCS (markedly L lateralizing with spinal shock)	Clinical pathology: PCV, TS, radiometer, haematology and biochemistry unremarkable MRI: Intramedullary focal lesion at the level of L1-2 IVD that is T2W hyperintense and T1W iso- to hypointense. At the same level, there is a left sided extradural non-compressive material, T2W hyperintense, T1W isointense. The NP at the level of the IVD L1-L2 has normal T2W signal but reduced in volume.	Diagnosis: ANNPE at the level of the IVD L1-L2	Medical tx: cage rest, physiotherapy and hydrotherapy while hospitalised for 3 d, continuation of rest and physiotherapy at home and at hydrotherapy center	4 w re-examination: improved L lateralizing ambulatory paraparesis and proprioceptive ataxia, increase controlled exercise, continuation of physiotherapy and hydrotherapy at home (last hydrotherapy after 12 months)

1. Case log organisation: This is entirely left to the candidate's judgement, and it is one of the assessment points. However, listing the cases in chronological order is preferred.

2. Localisation should reflect neurological examination findings, not the investigation findings. Multifocal cases should have further explanation (i.e.: left forebrain and brainstem)

3. Provide normal range for clin path results. Imaging description: rather than providing too much detail or just giving diagnosis, succinct description of lesion using correct terminology and signal changes on different sequences is more desired.

4	3 31/08/15, 87563m Feline, DSH, MN/1y4m	History: 1 m history of progressive non- ambulatory tetraparesis. Neurological examination: Non ambulatory tetraparesis, intact postural reactions when adequately supported. Reduced spinal reflexes in all limbs. Mildly decreased palpebral reflex on both eyes. Diffuse muscle atrophy of pelvic limbs	Neuromuscular, Peripheral nervous system	Clinical pathology: mildly elevated CK (841 u/l, RI 52- 506). Normal ACh receptor antibody titre. Thoracic radiographs: normal. Abdominal US: normal. Electrodiag: fibrillation potentials and positive sharp waves in the distal limb muscles. Decreased CMAP (2-3mV, n: 16.9-39.5)) in sciatic-peroneal nerve. Normal MNCV (100 m/s). Normal RNS. 5	Chronic polyneuropathy: suspected demyelinating and remyelinating polyneuropathy	Medical tx: Intensive physiotherapy, hand feeding and nursing care. Progressive deterioration over next 3 w prompted muscle (vastus lateralis and triceps) and peroneal nerve biopsy. Histopath: demyelination and T cell infiltration into intramuscular nerve branches and peroneal nerve. Marked variability in myofiber size with scattered atrophic fibers.	Continued home physiotherapy and nursing care. Very gradual improvement over subsequent 4 m. Regained ambulatory status after 12 w. 6
4	04/01/16, 250535, Horse, Thoroughbred, ME/11y	History: 6-12m weight loss and muscle loss over shoulders and hindquarters (box rested). Neurological examination: base narrow stance with fasciculations particularly of triceps and neck; paraparesis, prolonged recumbency. Severe generalized muscle atrophy. Shifting weight. Marked PL weakness on tail pull test.	Neuromuscular - Diffuse	Clinical pathology: moderately elevated AST (835U/L, RI 258-554U/L) and CK (654U/L, RI 150-385U/L), moderate neutrophilia ($10.863 \times 10^9/L$, RI 3.00-6.40 $\times 10^9/L$), markedly decreased Vit E (0.6mg/L, RI > 1.29 – 8.61mg/L), selenium (363nmol/L, RI 1200-3200nmol/L), and glutathione peroxidase (GSH-Px) (9/ml RBC, RI 15-600u/ml RBC) concentrations. Histopathology: Semimembranosus and sacrocaudalis dorsalis muscle: widespread and generalised angular atrophy with some fibres with internalised nuclei and endomysial cluster of mononuclear inflammatory cells. Some mononuclear cells were actively phagocytosing necrotic appearing fibres. Low amounts of appearing glycogen in most fibres.	Mixed degenerative myopathy and neuropathy (Equine motor neuron disease) - suspected vitamin E deficiency related	Medical tx: Vit E/Selenium	7d: less muscle fasciculation. Putting on weight. 2w: significant improvement in vitamin E concentrations, modest improvement in Selenium and GSH-Px. Improvement in CK and AST. Clinical improvement: reduced fasciculation, less time recumbent during the day and subjectively stronger on walking tail pull

4. Cases should be presented in a succinct manner (2-4 cases per page). If there are too many one page cases, case log will be returned to the candidate to shorten.

5. Always provide names of the tested nerves and normal reference ranges.

6. Provide adequate follow up. There is no specific time limit. This should be appropriate for the diagnosis.

5	12/04/16 18957 Canine, Whippet, ME/8m	History: 2 days of PUO and neck pain. Neurological examination: Mentation: QAR. Cranial nerves: normal. Proprioception: normal in all four limbs. Segmental reflexes: normal in all four limbs. Gait: Stiff gait with low head carriage. Palpation: cervical and lumbar hyperaesthesia.	Multifocal pain	Clinical pathology: WBC 20x10⁹/L (5.05-16.76) and neutrophils 16x10⁹/L (2.95-11.64). CRP: 189mg/L (n<10). MRI: STIR hyperintense and contrast enhancing longus colli muscle at the level of C4 vertebral body. CSF (cisternal): protein 0.62 g/l (<0.25) and NCC 287/ul (0-6) with prevalence to neutrophils. CSF PCR (Toxo & Neos): -ve	Steroid Responsive Meningitis Arteritis	Medical tx: Prednisolone (1mg/kg q12hr PO)	Weaned off steroids over 6 months, no sign of relapse 1 year after diagnosis.
6	10/9/16 18547 Canine, Labrador, MN/8y	History: Acute onset balance loss and difficulty walking. Neurological examination: Mentation: BAR, anxious. Cranial nerves: OU horizontal nystagmus, fast phase to left, right head tilt. Proprioception: normal in all four limbs. Segmental reflexes: normal in all four limbs. Gait: severe vestibular ataxia without paresis. Palpation: Not painful.	Cranial nerves: Right CN VIII	Clinical pathology: Bloods: normal CBC and biochemistry. Normal serum T4. MRI: Normal brain, middle and inner ear structures. CSF (cisternal): normal.	Idiopathic Vestibular Syndrome	None	Normal neurological examination at 4 weeks re-check apart from mild head tilt.
7	5/03/17 19635 Ferret, M.3y5m	History: Progressive hind limb weakness over the last 2 weeks. Developed urinary and faecal incontinence last 2 days. Neurological examination: Mentation: BAR. Cranial nerves: normal. Proprioception: normal hopping in forelimbs, absent in hind limbs. Segmental reflexes: reduced/absent in hind limbs. Gait: paraplegic with intact nociception. Palpation: not painful.	Spinal cord: L4-S3	Clinical pathology: elevated globulins 37 g/l (18-31). MRI: T2 and STIR hyperintense, noncontrast enhancing, ill defined, intramedullary lesion at the level of last thoracic vertebrae. CSF (lumbar): protein 0.75 g/l (0.28-0.68, NCC 898/ul (0-8) with predominance to lymphocytes. PCR for Distemper and Toxoplasma negative. Normal protein electrophoresis. Aleutian disease negative. Serum Toxoplasma IgM <20, IgG positive at 1:152.	Meningomyelitis	Medical tx: Dexamethasone (iv 0.2mg/kg q24hr)	Euthanised after 48 hours because of loss of nociception. No PM performed.

8	17/05/17, 10537830, Equine, Connemara, Gelding/12y	History: 2 w Hx of progressive R hindlimb lameness. No response to anti-inflammatory medication. Hoof abscess ruled out by RVS. Neurological examination: Grade 4/5 R PL lameness, hypometric R PL, unable to fully support weight through the R PL due to inability to extend the stifle, atrophy of the quadriceps mm., normal spinal reflexes & cranial nerves, tail pull & other tests of gait not performed for safety reasons. Soft tissue mass palpable within epaxial musculature in the R caudal lumbar region.	Neuromuscular (peripheral neuropathy): R femoral nerve (L4-L6)	Imaging: Scintigraphy: large, circular, well defined area of increased radiopharmaceutical uptake in R paralumbar region; Ultrasound: well defined, encapsulated 12cm ovoid mass with complex echogenicity, moderate vascularisation abutting the lumbar transverse processes. Post-mortem 3T MRI (LS): complex, extensive & multilobulated T1-W hyperintense mass lesion involving the paravertebral soft tissue from caudal L2 to level of L5 with invasion of the L3-L4-L5 R intervertebral foramen into the vertebral canal causing R ventral extradural compression of the SC. Histopath.: ultrasound guided Tru-cut biopsy: consistent with melanoma.	L3-L5 paraspinal melanoma.	Euthanasia.	Euthanasia.
9	28/08/17, 10518485, Equine, Warmblood, FE/10m	History: 3d history of diarrhoea & inappetence progressing to somnolence, head pressing & ataxia. Neurological examination: stuporous mentation with intermittent periods of hyperactivity, low head carriage, PC tetra-ataxia, tendency to circle, absent menace response bilaterally.	Brain – forebrain (cerebral cortex).	Clinical pathology: BHE: profound neutropenia ($0.04 \times 10^9/L$, RI 0.00-0.10 x $10^9/L$), hypoproteinaemia (albumin 22.8g/L, RI 23-35g/L, globulin 18g/L, RI 30-50g/L), azotaemia (creatinine 242μmol/L, RI 22-134μmol/L, urea 8.5mmol/L, RI 2.5-8.3mmol/L), ammonia high end of reference interval (85μmol/L, RI 15.00-90μmol/L), elevated serum amyloid a (237.7mg/L, RI 0-20mg/L); faecal culture: <i>Lawsonia intracellularis</i>. Imaging: AUS: small intestinal thickening with distended stomach.	Encephalopathy due to <i>L. intracellularis</i> infection causing dysbiosis & hyperammonaemia.	Medical tx: IV antibiotics (oxytetracycline), NSAIDs, IVFT, supportive care, dexamethasone.	Significant improvement in mentation with medical treatment. Discharged after 7 d hospitalisation. Represented for L PL lameness 3 m later. No neurological deficits detected at this point.

10	26/10/17, 10527585, Lupine, Giant Continental, Rabbit, ME/1	History: Progressive paraparesis, gut stasis & weight loss. Neurological examination: Paraplegic with absent hopping in the PLs, increased patellar reflex & normal withdrawal reflex bilaterally, perineal reflex intact, possible hyperaesthesia on palpation of the thoracolumbar vertebral column, DPP in the PLs difficult to interpret.	Spinal cord: T3-L3 SCS	Clinical pathology: BHE: leukocytosis ($4.71 \times 10^9/L$, RI 5.20-$12.50 \times 10^9/L$) with mildly toxic neutrophils on blood smear evaluation, elevated CK (1453U/L, RI 59-175U/L) & globulins (34.8g/L, RO 15-33U/L); <i>Encephalitozoon cuniculi</i> IgM: positive 1:80, IgG: positive 1:1280; Bacterial C+S on PM sample: <i>E. faecium</i> <i>E. casseliflavus</i> & <i>S. lugdunensis</i>. CT (mousetrap): narrowing of the T10-11 & L3-4 IVD spaces with patchy, multifocal lytic lesions & irregular periosteal reaction involving the affecting vertebral end plates; focal hyperattenuating & CE material within the ventral aspect of the vertebral canal at the level of T10/11 causing moderate SC compression. Histopath. (PM): chronic, suppurative discospondylitis with intervertebral abscessation (L1-2-3-4, T10-11).	Multifocal discospondylitis with secondary empyema. Concurrent <i>E. cuniculi</i> infection.	Medical tx: Analgesia (meloxicam, gabapentin, paracetamol), antibiotics (metronidazole, enrofloxacin, doxycycline), supportive care (metoclopramide, cisapride, biolapis & oxbow critical care).	Euthanasia 4 w after diagnosis due to lack of neurological improvement.

Case examples in the following pages are poorly written cases. Significant number of similar mistakes will lead to case log for failure or request for revision.

11	10/11/17 19745 Canine, French bulldog, MN, 4y	History: peracute onset of inability to walk after jumping from the sofa. Neurological examination: Paresis and proprioceptive ataxia of the PL. Reduced postural reactions RPL, absent LPL. Reduced withdrawal reflexes PL. Painful at T13-L1	Spinal cord: T3-S3 SCS	MRI: intervertebral disc extrusion L5-6.	IVDE L5-6	Surgical: Right sided L3-4 Hemilaminectomy (primary surgeon)	Normal neurological examination at 4 week recheck
12	10/01/18 19835 Canine, Cocker Spaniel, FN, 9y	History: Subacute progressive history of loss of balance to the left side and left corneal ulcer. Neurological examination Mentation: BAR. Posture: left head tilt Gait: left vestibular ataxia Proprioception: normal. Cranial nerves: spontaneous nystagmus rotatory fast phase to the right, absent palpebral reflex OS. Segmental reflexes: normal Palpation: not painful.	Brain – L brainstem (medulla) Correct NAL: Cranial nerves: Left CN VII + CN VIII	MRI brain: single, ill-defined, intra-axial lesion located in the left medulla oblongata that is T2W/FLAIR hyperintense, T1W isointense and mild/moderate CE. No mass effect or perilesional oedema. CSF (cisternal): mild mononuclear pleocytosis (TNCC 25/ul, protein 31.7mg/dl [$<25\text{mg/dl}$]). Neospora serology: negative	Meningoencephalitis of unknown origin	Medical tx: Tapering dose of prednisolone (from 1mg/kg BID)	3d after discharge: slight improvement in clinical signs. 2m: complete resolution of clinical signs. Weaned off prednisolone over 5 months. No sign of relapse at last phone check up 12 months from diagnosis.

Case 11: Incomplete neurological examination, vague neuroanatomical localisation and lack of MRI definition of the pathology. Cutaneous trunci reflex should be reported to aid localisation. Neuroanatomical localisation should explain the uncertainty (i.e.: T3-L3 SCS with spinal shock vs L4-S3 SCS). MRI findings should give the detailed findings rather than imaging diagnosis.

Case 12: Neuroanatomical localisation should be the conclusion of neurological examination, rather than conclusion of investigations. This is a common recurring error in case logs received by the committee.

13	11/01/18 19865 Canine, French Bulldog, MN, 5y	History: Acute onset paraparesis. Neurological examination: Non-ambulatory paraparesis. Proprioception: absent in pelvic limbs, normal in thoracic limbs. Cranial nerves: OU absent menace response, vision and PLRs. with normal palpebral reflexes and intact vision/tracking. Palpation: severe thoracolumbar hyperaesthesia	Spinal cord: T3–L3 SCS	MRI spine: L2–L3 IVDE with moderate spinal cord compression.	L2-3 IVDE	Surgical: L2–L3 hemilaminectomy (primary surgeon)	Recovered ambulation at 4 weeks.
14	01/02/18 20105 Canine, cross breed, MN, 3y	History: 2 days of neck pain. Neurological examination: Mentation: QAR. Cranial nerves: normal. Proprioception: normal in all four limbs. Segmental reflexes: normal in all four limbs. Gait: Low head carriage with normal gait. Palpation: cervical hyperaesthesia.	Spinal cord: C1-5 SCS	Clinical pathology: Bloods: normal CBC and biochemistry. MRI: C3-C4 intervertebral disc space is narrowed with large amount of T2 hypointense material along the ventral aspect of the spinal cord from cranial C3 to caudal C4	C3-4 IVDE	Surgical: C3-4 ventral slot (primary surgeon)	Discharged 2 days after surgery with no cervical pain and normal neurological examination. Normal on 4 weeks re-examination.

Case 13: Incomplete NAL: The stated localisation ignores the recorded cranial nerve deficit without explanation. It must either be localised as Multifocal, or the cranial finding must be explicitly justified, e.g.: "Spinal cord: T3–L3 SCS (bilateral absent menace response secondary to ocular pathology) or Multifocal: T3-L3 SCS and bilateral optic nerves). MRI findings should give the detailed findings rather than imaging diagnosis.

Case 14: Wrong NAL: This is a common error. NAL is given to match investigation findings; NAL should reflect neurological examination findings. Correct NAL is: Hyperaesthesia without neurological deficits (cervical meninges, nerve roots, facet joints, IVD, vertebrae, muscles).

