

Satellite Article

Polyuria-polydipsia in the horse

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Introduction

Polydipsia-polyuria (PU/PD) in the horse is a rare but important clinical sign usually indicating a failure of normal homeostatic mechanisms controlling water balance. It can be caused by increases in water intake or urinary output and the primary cause is the key to possible treatment. Failure to address the underlying causes for the complex of signs and application of symptomatic treatment alone may be both disappointing and, in some cases, harmful. Although it is easily interpreted as being a major pathological event, it is important to realise that there are physiological reasons both for the increase in drinking and the passage of larger than normal volumes of urine. **Establishing whether a horse is primarily polyuric or polydipsic is not always easy, especially in horses kept at pasture.** Often owners mistake **pollakiuria** (increased frequency of urination) for increased volume of urination (**polyuria**). Even when the volume of drinking can be measured accurately (such as in a stall/stable) the presence of increased consumption may be either a primary polyuria (with consequent polydipsia) or a primary polydipsia (with consequent polyuria). The presence of a 'wet stable' may be suggestive of a PU/PD problem, but again this is a very subjective assessment method! It may be necessary to collect 12 or 24 h urine output and this itself is not an easy task requiring the use of purpose made collection devices that are sometimes not well tolerated.

Browning (2000) reports on polyuria and polydipsia in 2 horses caused by psychogenic polydipsia. **In this issue, a brief review of the physiology forming the background to the condition is described, together with the diagnostic and differential diagnostic approaches.**

A brief review of renal physiology, urine production and water homeostasis

Water homeostasis in the normal horse is achieved through a balance of water consumption, metabolism, excretion and insensible losses (Roussel and Carter 1989) (Fig 1). **Drinking** and **urination** are the main routes by which fine control of this balance is achieved (Brown 1997).

Water intake

Water is added to the body fluid compartments by one of 3 mechanisms:

- **Through drinking.** The thirst centre in the hypothalamus controls this. Not all the water intake is derived from drinking - indeed, in the horse, the majority of water intake is derived from other sources (see below). Water intake via drinking is regulated to ensure that the balance is maintained and if extra or less water is therefore obtained from the other sources, the thirst responses will be correspondingly adjusted. This may explain the apparent 'lack of thirst' in horses grazing in early spring and summer, and in horses fed moist or succulent diets.
- **Through food.** Ingestion of food is directly responsible for the intake of a large proportion of the water requirements of a horse but the actual supply depends on the nature of the food. A well-soaked net of hay may provide a considerable volume of water while a diet high in dry content obviously provides much less. The actual amount of water supplied by the diet can be calculated fairly accurately from dry matter analysis of each component.
- **Through metabolism.** Up to 10% of the water needs can be provided by the metabolic breakdown of carbohydrates, fats and proteins (Roussel *et al.* 1989). There is no difference in metabolic terms between this water and water derived from drinking or food. The actual amount of water the horse derives from its metabolic processes tends to be fairly constant, but is actually hard to quantify.

Water maintenance requirements have been estimated at 40–60 ml/kg bwt/day (Freestone *et al.* 1995). Water requirements are, in fact, proportional to metabolic body size rather than weight and, therefore, a Shire horse requires relatively less water/kg bwt than a Shetland pony. Furthermore, fat animals also appear to require less water than lean ones, possibly due to the low level of water in body fat.

Water losses (Roussel *et al.* 1989; Brown 1997)

Water loss occurs through various mechanisms including:

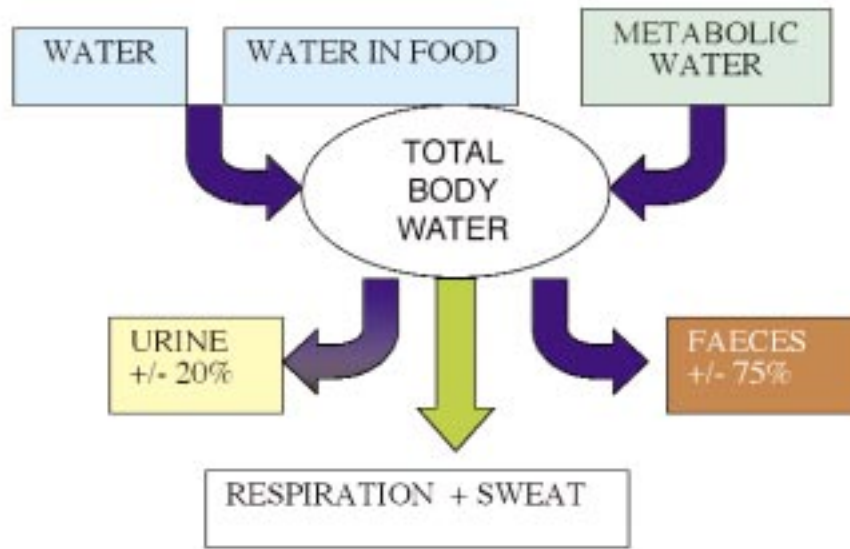


Fig 1: Water homeostasis.

- **Urine.** Although urination is a very visible means of fluid loss it is not the major loss. The volume of urine produced is very variable and acts as a fine overall controlling mechanism. Between 15 and 30 ml/kg bwt/day of urine are produced.
- **Faeces.** Water loss through the faeces is the major loss and accounts for 75% of the total.
- **Sweat, insensible skin loss and water vapour in expired air.** Losses in this category are variable and depend heavily on work and environmental factors. There is a linear relationship between water intake and environmental temperature (Haupt 1987).
- **Pathological loss.** This includes blood loss, sweating, diarrhoea and serum exudation.

In addition, dietary composition has been shown to affect the amount of urine produced. Pelleted feeds and legume hay produce more urinary water than grass hay due to the higher concentration of renal solutes. As a general guide, it has been estimated that 2–4 l water/kg dry matter consumed/day are required to maintain homeostasis (Haupt 1987).

Water homeostasis

Maintenance of water balance depends upon establishing a balance between water intake and excretion, so that plasma osmolarity is maintained within 2% of normal. As a horse becomes water depleted, both osmolarity and concentration of sodium ions in the plasma increase. **This produces a positive direct feedback mechanism onto osmoreceptor cells** in the rostral diencephalon and an indirect feedback from arterial and left atrial baroreceptors, via the vagus and glossopharyngeal nerves.

The net effect is the release of stored antidiuretic

hormone (ADH) from the neurohypophysis of the pituitary gland (Roussel *et al.* 1989; Freestone *et al.* 1995). ADH is a polypeptide that is synthesised in the suprachiasmatic, paraventricular and supraoptic nuclei of the hypothalamus. It is transported via secretory granules, from the latter 2, to the neurohypophysis where it is stored. The suprachiasmatic nuclei distributes its ADH to other areas of the CNS via a neuronal distribution network. As plasma osmolarity rises, stored ADH is released from the neurohypophysis and acts on the collecting tubules of the kidney, where it causes increased uptake of water and solutes, therefore increasing the specific gravity of the urine.

Water diuresis occurs whenever the osmotic pressure of the plasma is reduced to a level that will not stimulate ADH production (usually a result of excessive water intake). Excess substances other than water must be kept in solution or they cannot be excreted and this produces an osmotic diuresis. The water required to act as a solvent produces increased urine volume.

Urine is the product of 2 major processes: **blood filtration and modification of the resulting filtrate.** High pressure modified capillaries in the renal glomerulus filter out large volumes of water and small solutes, separating them from larger molecules such as proteins that remain in the capillary lumen. This **filtration barrier** against the larger molecules works on the basis of variations in shape, electrical charge and size. The volume of plasma filtered in a given time is termed the **glomerular filtration rate (GFR)** and is approximately 20% of the plasma volume delivered to the kidneys.

1) Over 98% of the water in the glomerular filtrate is reabsorbed in the renal tubules and collecting ducts (**Fig 2**). If the entire glomerular filtrate were lost this

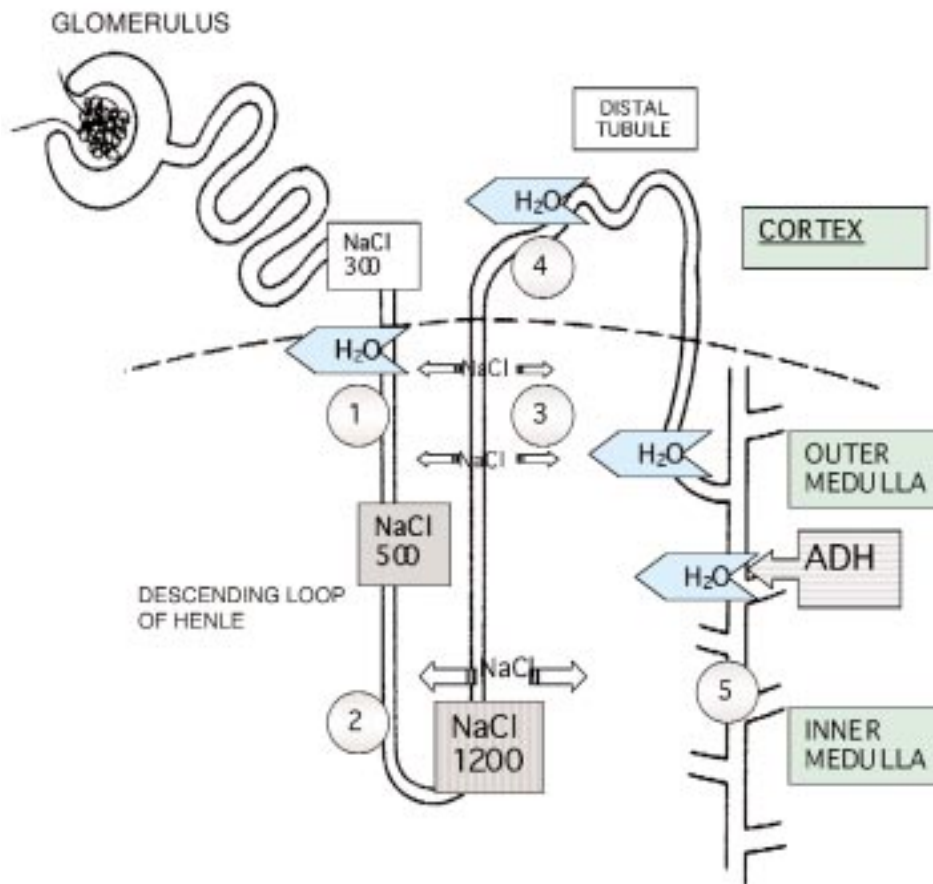


Fig 2: Water recovery in the equine kidney.

would represent a totally unacceptable loss of water and small solutes. **Modification of the filtrate** therefore occurs in the renal tubules, which recover large volumes of water and solutes. Much of the physiological mechanism for reabsorption is energy demanding.

Urine output varies from 5–15 l/day and the **specific gravity** of the urine will vary between 1.020 and 1.050. Urine production is a mechanism that relies heavily on:

- i) the **maintenance** of renal interstitial hypertonicity;
- ii) the **action** of ADH on Na^+ by electrostatic attraction,
- iii) water **impermeability** in the absence of ADH.

These factors help to maintain interstitial hypertonicity and produce a dilute filtrate.

2) The glomerulus is a barrier to large molecules (**Fig 2**). It is around 100 times more permeable than normal capillary membranes and acts a sieve. Molecules less than MW 65,000 pass readily into the filtrate while larger ones, such as fats and colloidal proteins, and cells, are retained. The filtrate has, therefore, approximately the same osmolarity as plasma except that it contains small amounts of protein (and no cells).

One of the most significant filtrates is glucose, which passes readily into the filtrate. Normally the concentration in the blood is restored by complete reabsorption in the proximal tubules. If the transport

capacity is exceeded (tubular maximum), glucose remains in the filtrate and carries water with it. Therefore the urine contains glucose and will be more dilute (such as is the case in true *diabetes mellitus* and in Cushing's disease in horses). Loss of the active reabsorptive mechanism as a result of tubular disease might permit glucose to pass into the distal tubules and so present with glycosuria and polyuria.

3) Water is absorbed in the distal tubule by osmosis (**Fig 2**). The **collecting tubules** are subject to a high osmotic gradient but are only permeable to water in the presence of ADH. Over 80% of the glomerular filtrate has been absorbed by the time it reaches the distal tubule and, therefore, only 20% is subject to the action of ADH.

4) Water is reabsorbed along the osmotic gradient of the descending loop of Henle, which is permeable to water but impermeable to solutes.

5) The resultant concentration of NaCl results in a hypertonic solution. The distal end of the loop and the thin portion of the ascending Loop of Henle are permeable to solutes but not to water. This allows passive diffusion along a concentration gradient of NaCl into the interstitium (**Fig 2**).

In the thick part of the ascending loop, active transport of Cl^- and the accompanying uptake of Na^+ by electrostatic attraction, plus water impermeability help to maintain interstitial hypertonicity and produce a dilute filtrate (**Fig 2**).

A second hormonal mechanism involved in water regulation is the **renin-angiotensin system**. During hypovolaemia, renin is released from the juxtaglomerular cells of the kidney. It acts on renin substrate, converting it to **angiotensin I**, which is immediately converted to **angiotensin II**. This compound has a direct effect upon renal blood flow, causing water conservation, and an indirect role by stimulating the production of **aldosterone** from the adrenal cortex. Aldosterone has a direct action on the renal tubules, which causes the conservation of sodium ions and water and the selective excretion of potassium ions. The sodium sparing effects of aldosterone are not just limited to the kidney. The equine diet is relatively rich in potassium and low in sodium.

Aldosterone levels also rise in response to exercise and eating and have been shown to cause the selective uptake of sodium from the colon, thereby reducing the level of postprandial hyperkalaemia. This is, therefore, an important mechanism in maintaining sodium levels after feeding and exercise and therefore the osmolarity of the extracellular fluid (Harris 1993).

When these mechanisms are overcome, drinking is induced through the stimulation of **receptors in the oropharynx**, which detect dryness of the mouth and controlled by osmosensitive neurones in the hypothalamus (Kohn and Hansen 1998). The complex of mechanisms that adjust the water and electrolyte balances within the milieu interieur is in exquisite balance. It is true to say that the "dumbest kidney is cleverer than the cleverest clinician" when it comes to the control of water loss via the kidneys.

The horse produces alkaline urine with a pH of around 7.6–8.5. Adult equine urine normally contains high concentrations of calcium salts (usually carbonate). This is often saturated and so a cloudy, yellow-white particulate precipitate can usually be seen in normal horse urine. High dietary oxalate can result in oxalate crystals. Concurrent infection or a nidus formed by some other mechanism can result in the development of large urinary (renal, ureteral and cystic calculi). Renal failure, by contrast to many other species, results in the failure of the normally high calcium excretion via urine. This results in water-clear urine and raised serum calcium concentration, which is not a result of secondary hyperparathyroidism. Although this is characteristic of renal failure, its extent depends strongly on the amount of calcium in the diet (King and Schott 1997).

Furthermore, the renal pelvis contains a high number of mucus-secreting cells. This results in the presence of protein in normal horse urine and often imparts a mucoid appearance to the normal flow of urine. Renal failure, or the production of large volumes of urine for any other cause, dilutes the mucous component and so the urine loses its mucoid appearance.

Differential diagnoses of polyuria-polydipsia

PU/PD occurs when there is abnormal thirst or urination. Many disease processes have been implicated in this syndrome. **The major decision that faces a clinician is to decide whether the problem is related to pathological polyuria or to polydipsia.** This decision is fundamental to the subsequent investigations, but it can be very difficult to establish which option exists.

- **A horse with diarrhoea** may produce less urine than normal but drink more than normal.
- **A horse that ingests a high salt diet** drinks more than normal and, as a consequence, produces larger volumes of urine.
- **A horse with renal failure** that is unable to concentrate urine produces more urine and will therefore need to drink more.

The flow chart shown in Figure 3 outlines a general approach that can be used to establish the major causes of the PU/PD clinical sign.

Polydipsia

Psychogenic polydipsia

Remarkably, this is one of the commonest causes of PU/PD in mature stabled horses (Brown 1997). It is often seen in young animals (Freestone *et al.* 1995) and is usually considered to be a vice associated with boredom (Roussel *et al.* 1989; Freestone *et al.* 1995; Brown 1997). The horses have often had a change in management or environment that is conducive to stress or boredom. Dietary factors have also been implicated as a cause, including static routine feeding times, high dry matter intake (Houpt 1987) and excessive (or compulsive) salt intake (Buntain *et al.* 1981). Affected animals often eat their bedding or chew on doors, etc. This syndrome is described in detail in a case report by Browning (2000).

The PU/PD associated with psychogenic polydipsia is often more severe than that seen with **chronic renal failure (CRF)** or **equine Cushing's disease (ECD)** (Brown 1997). The animals are usually in good body condition and have no biochemical evidence of renal failure. The urine is invariably very dilute with a low specific gravity ($\text{SG} < 1.005$). Cases with a short history of PU/PD are commonly able to concentrate their urine in response to water deprivation while longer-standing cases may have no such ability. It is suggested that, in the latter cases, the inability to concentrate urine is due to renal 'medullary wash-out' in which the normal osmotic gradients between the tubular lumen and the renal interstitium are reduced to the point where urinary concentration cannot take place (Roussel *et al.* 1989; Brown 1997). **In spite of high levels of antidiuretic hormone (ADH)** (natural or administered) **the tubule fails to respond by increasing the specific gravity (SG) of the urine.** Initially, the horse

can be subjected to a water deprivation test whereby all water is removed for 12–24 h (see below).

- **It is important to note** that although psychogenic polydipsia is a common disorder, total water deprivation should not be performed before renal function is tested.

If this fails to induce urinary concentration, a modified water deprivation test should be performed (see below). This test is performed over 2–4 days and by the end of the test the horse with renal medullary washout will have restored the gradients and produce urine with SG>1.025.

The management of psychogenic polydipsia is described by Browning (2000) but can be summarised as:

- **Alteration of management system (including feed, bedding, routine), environment (turn-out, walker etc.) and exercise regimen** (Brown 1997; Browning 2000).
- **Limitation in water intake through restriction over 2–4 days** (Roussel *et al.* 1987; Brown 1997; Browning 2000).
- **Electrolyte therapy:** Sharp (1980) found that broad electrolyte replacement was effective in treating 5 cases of PU/PD. This management is described in the accompanying paper by Browning (2000).

Thirst

High ambient temperature and humidity usually induce thirst, particularly if this is accompanied by ingestion of dry foods. Loss through sweat and via the respiratory system represent significant demands on the water status of the horse and drinking needs to compensate for these. Interestingly, high environmental humidity may also increase thirst.

Compulsive salt/glucose consumption

Compulsive salt or glucose consumption is a rare event. Usually these horses ‘attack’ a salt or glucose block - often ingesting large chunks of the solid material. The clinical history is invariably clear, although the reasons behind the habit are usually much less so. There is often some consequent diarrhoea (possibly as a result of osmotic retention of fluid in the intestinal lumen) and, possibly, even severe intestinal derangement. These serve to increase the thirst still further. In the case of salt ingestion, electrolyte analysis reveals a very high sodium clearance ratio (see below). Glucose ingestion in sufficient quantity might be expected to have a more profound effect in inducing a dramatic change in the large colon function and pH. Although such animals might drink excessively, there are invariably more dramatic signs of endotoxaemia and diarrhoea.

Polyuria

Polyuria can be induced primarily by a number of pathological processes and secondarily through the

action of other pathological processes in the body. Urine production is a vital body function and, even in the face of extreme water deprivation, urine production will continue. It is most unusual for any animal to be so deprived of water that only the obligatory minimum volume of urine is produced. The urinary output is the only measure of fluid loss that can be quantified in a practical situation.

Diabetes mellitus (Freestone *et al.* 1995; Whitlock 1992; Brown 1997)

True pancreatic diabetes mellitus is extremely rare but has been described as a result of verminous pancreatitis (Bulgin and Anderson 1983). Polyuria is caused by osmotic diuresis induced by the glycosuria associated with this condition. However, **by far the majority of horses exhibiting hyperglycaemia and glycosuria** have dysfunction of the *pars intermedia* of the pituitary gland variously termed equine Cushing’s disease (ECD) and secondary hyperadrenocorticism (Merritt 1987) (see below).

Diabetes insipidus (Freestone *et al.* 1995; Whitlock 1992; Brown 1997)

This can arise either from:

- **failure of production of antidiuretic hormone** (ADH) from the *pars distalis* of the pituitary gland (central *diabetes insipidus*) (CDI). CDI has been reported following viral encephalomyelitis or from compression of the *pars distalis* of the pituitary gland as part of equine Cushing’s disease (ECD) syndrome (Brown 1997).
- **lack of tubular response to ADH** (nephrogenic *diabetes insipidus*) (NDI). NDI has been reported as a familial condition (Schott *et al.* 1993) and secondarily to acute renal bacterial infections (Roussel *et al.* 1989).

Hyperadrenocorticism (Freestone *et al.* 1995; Whitlock 1992; Brown 1997)

Dysfunction of the *pars intermedia* (PI) in equine Cushing’s disease is by far the commonest cause of adrenal cortical hyperplasia. The hyperplastic *pars intermedia* results in excessive pituitary production of adrenocorticotrophic hormone (ACTH) (Orth *et al.* 1982). This, with the steroidogenic effects of melanophore-stimulating hormone (MSH) and corticotropin-like intermediate peptide (CLIP), results in the characteristic hypercortisolaemia. This results directly in increased glomerular filtration rate and secondarily to hyperglycaemia, both of which induce polyuria (Love 1993).

Sepsis and endotoxaemia

Prostaglandin E₂ produced in response to endotoxin has a powerful vasodilator effect on the renal blood vessels and also reduces the sensitivity of renal collecting tubules to ADH (Whitlock 1992).

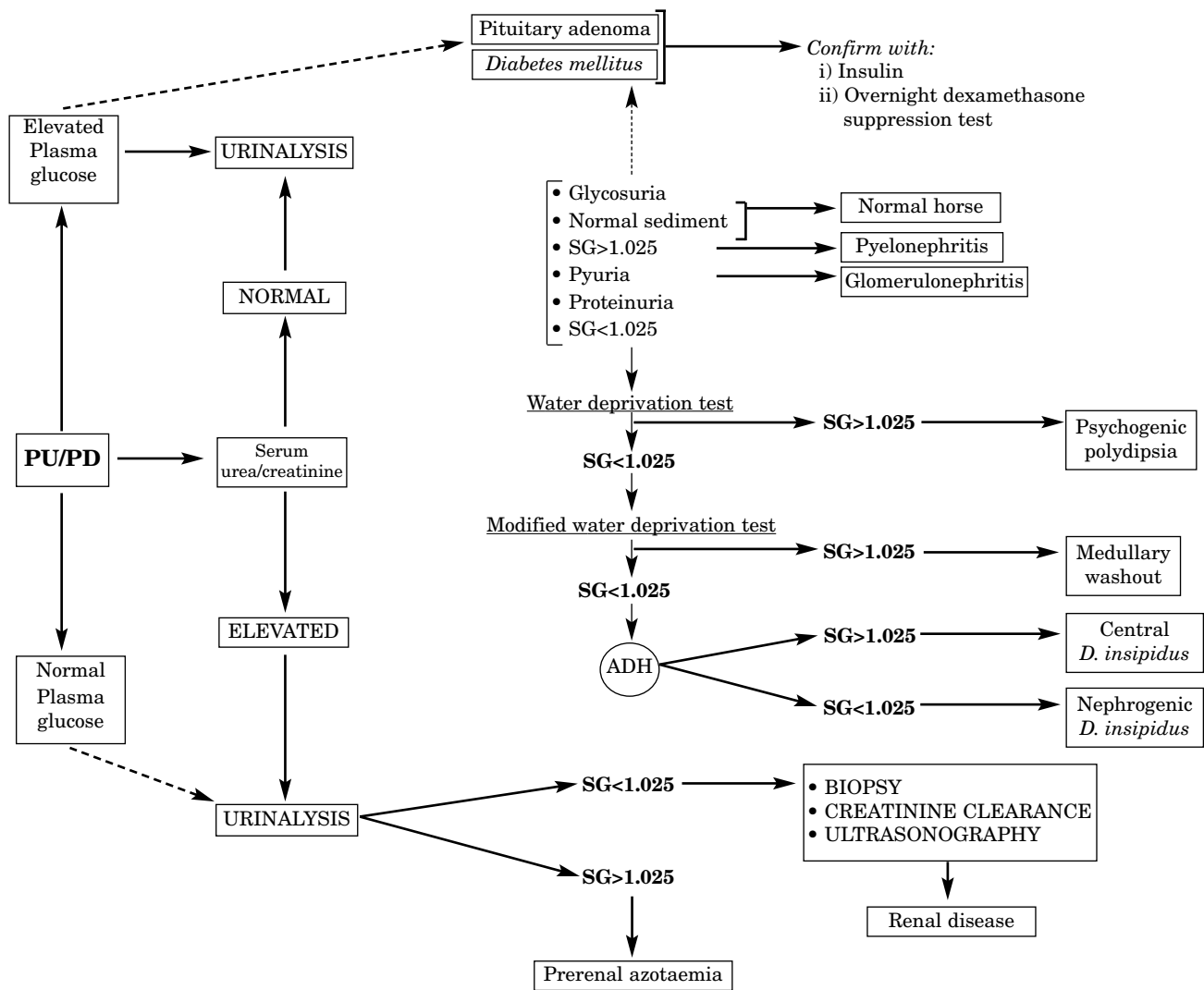


Fig 3: Investigation and diagnostic protocol for suspected polydipsia/polyuria.
 Note: This figure illustrates only the common causes and it is wise to remember that some more complex syndromes may be found! Also note that the blood glucose estimations can sometimes be misleading for other physiological reasons (after Brown [1997]).

Iatrogenic

Sedation with α_2 agents, corticosteroid administration and diuretics all have direct effects on urine production (Brown 1997; Buntain and Coffman 1981). The mechanisms for each of these is somewhat different but they all exert their effects on the kidney and are therefore primary causes of polyuria. A consequent polydipsia may be detected subsequently. Intravenous fluids result almost inevitably in a profound diuresis and this is especially marked if the flows are nonphysiological. This is not usually accompanied by a polyuria because it is due to water overload rather than electrolyte lead diuresis.

Primary renal insufficiency or failure

This is frequently implicated as a cause of PU/PD, although the incidence in clinical practice is low and only

33% of cases of CRF demonstrate PU/PD. Theoretically the extent of PU/PD depends on the extent of tubular damage but the severity of PU/PD is usually much less than would be expected (King and Schott 1997).

Diagnostic approach in PU/PD (Fig 3)

History

The duration of the problem should be established and related to changes in management or feeding. Any previous medical history, such as atypical laminitis or failure to shed hair in the spring and summer, should be noted.

Clinical examination

A full clinical examination should be performed.

Variations in cardinal signs and abnormal auscultation findings may indicate underlying infectious diseases, which could be associated with **sepsis** or **ECD**.

Evidence of **supraorbital fat deposition**, **hirsutism**, current or chronic **laminitis**, pendulous abdomen should alert one to the possibility of **ECD** being implicated in the disease.

Hydration status should be evaluated as any existent azotaemia needs to be evaluated in the light of the hydration status and normal hydration needs to be established before a water deprivation test is performed.

Establish if polydipsia/polyuria is actually present

It is important to establish that the animal is PU/PD (**Fig 3**). **Many owners report that the animal is polyuric, when they are in fact pollakiuric.** This may be caused by cystitis, urethritis, vaginitis, metritis and oestrous behaviour. Excessive stall-wetting may be caused by the animal playing with its water buckets (Roussell *et al.* 1989). To establish, therefore, if the animal is PU/PD, it must be confined to its stall for 24 h. During this period, urine outflow can be measured using collection devices (Roussell *et al.* 1989; Brown 1997). A normal horse will produce 5–15 l urine a day (Brown 1997). More conveniently, water intake can be measured. If excessive water intake is measured, a full haematology, routine biochemistry and urine analysis should be performed. The urine should ideally be freely voided. If catheterisation has to be performed, α_2 agonists should be avoided because they can cause diuresis with reduction of urinary specific gravity and glycosuria.

Initial clinical pathology

If **hyperglycaemia** or **glycosuria** are present, then hyperadrenocorticism or *diabetes mellitus* should be suspected and appropriate testing for dysfunction of the pituitary gland should be carried out, e.g. an overnight dexamethasone suppression test or thyrotropin-releasing hormone stimulation test (Love 1993).

The detection of chronic inflammation on haematology, azotaemia and isosthenuria, especially in the presence of hypercalcaemia and hypophosphataemia (Brobst *et al.* 1978), may indicate the presence of CRF. The presence of leucocytes in the urine may further indicate the presence of pyelonephritis. It should be noted that 75% of the functional nephrons need to be lost before azotaemia in a normally hydrated horse occurs.

Renal function tests

The fractional excretion (FE) test has been used in the assessment of renal function (Harris and Gray 1992). The FE test may be used as part of the diagnostic evaluation of an animal with a suspected renal problem but abnormalities should not be used as the sole criteria for the diagnosis of renal compromise. Although definitive

information is not available, in a horse that has a diet with a normal sodium content and is not on i.v. fluids, **primary renal dysfunction is suggested by a sodium FE of >1%. Phosphate FE greater than 1%** may be suggestive of renal failure, but **excessive P intakes or a diet with an imbalanced Ca:P ratio** can result in markedly elevated FE PO_4 values in animals with normal renal function.

Water deprivation test

In the absence of azotaemia, dehydration or hyperglycaemia, one must differentiate between central and nephrogenic *diabetes insipidus* and psychogenic polydipsia. A water deprivation test assesses the ability of the renal tubules to react to ADH. A predeprivation urine sample is obtained and the horse is weighed. Water is withdrawn and urine samples collected frequently. The test continues until the SG of the urine exceeds 1.025, or the horse loses 5% of its bodyweight (Freestone *et al.* 1995).

In the field, it is not always possible to obtain such accurate bodyweights. This author routinely runs the test overnight, assessing dehydration status in the morning with packed cell volume and plasma protein estimation. If there are minimal signs of dehydration ($\leq 5\%$), the test continues until the urine has concentrated, or 24 h have elapsed. **If urine becomes concentrated to greater than 1.025, psychogenic polydipsia is confirmed.**

Modified water deprivation test

If concentration fails to occur, a modified water deprivation test is performed, where the water is restricted to 40 ml/kg bwt a day, which should result in urinary concentration, even in cases of psychogenic polydipsia with medullary wash-out (Brown 1997). An alternative method is to apply the standard water deprivation test, after i.v. administration of hypertonic saline (Roussell *et al.* 1989).

ADH stimulation test

If, after 4 days, the urine still will not concentrate, one is likely to be dealing with a case of *diabetes insipidus*. It is possible to differentiate between the central and nephrogenic forms of the disease by injecting 60 iu synthetic ADH i.m. every 6 h. If the urine concentrates, the diabetes is central; if it does not, it is likely to be nephrogenic (Brown 1997).

Other tests and investigations

- **Urinary tract ultrasonography is a very valuable tool but it does require experience in interpretation** (Reef 1998). In chronic renal failure, the kidneys are usually somewhat smaller than normal. The cortices are obviously narrowed with a higher echogenicity than normal and there is poor tissue differentiation of the internal architecture. An irregular contour can usually be appreciated with areas of high echogenicity where crystalline deposits are accumulated.

- **Endoscopy** (possibly in conjunction with individual sampling of urine from each ureter) is becoming increasingly feasible even in male horses (Schott and Varner 1996). It is important, however, to remember that suction and insufflation functions are virtually essential for cystoscopy.
- **Renal biopsy** is also entirely feasible and may help to confirm a diagnosis of renal disease in a case exhibiting PU/PD, particularly if the biochemical and other tests are supportive of the presumptive diagnosis. Renal disease is an infrequent diagnosis in the UK but it may be that it is more common than we realise. **Biopsy is not difficult and, with experience, the procedure has few hazards.** It should, of course, always be accompanied by ultrasonography.
- **Clearance tests.**

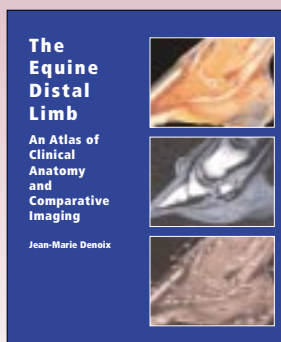
In summary, PU/PD is an interesting clinical challenge. Providing that a methodical and systematic approach is employed to diagnosis, it is usually possible to arrive at a definitive diagnosis. Although there are cases that can be treated with success, renal disease is probably best regarded as untreatable. Therefore, faced with a case exhibiting PU/PD, the clinician should pursue all diagnostic means possible to reach a definitive diagnosis and therefore a realistic prognosis.

Acknowledgement

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The Equine Distal Limb

An Atlas of Clinical Anatomy and Comparative Imaging

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Jean-Marie Denoix is the world's leading equine musculoskeletal system anatomist and has become one of the foremost equine diagnostic ultrasonographers. There is therefore nobody better to compile a reference atlas of the clinical anatomy of the foot, pastern and fetlock, correlated with images obtained by radiography, diagnostic ultrasonography and magnetic resonance imaging. Advanced imaging techniques require in

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