Finding a Formula

In clinical practice, serum osmolality is often used to evaluate patients. The osmole gap is determined by subtracting the calculated osmolality from the measured osmolality. In a healthy dog, osmolality is mostly determined by Na+, K+, Cl-, HCO3-, glucose, and urea in serum; the expected osmole gap in dogs is <10 mOsm/kg. Increases in the osmole gap can occur with some intoxications (eg, ethylene glycol, propylene glycol, ethanol).

In this prospective study, 18 previously published formulae for calculating osmolality were evaluated to determine the reference range for the osmole gap for each and to determine which formulae were most accurate regardless of the patient's BUN or glycemic status. Of the 250 study dogs, 30 were hyperglycemic, 22 were azotemic, and 200 were neither; 2 dogs were both hyperglycemic and azotemic. Serum osmolality was measured using an Advanced Micro Osmometer 3300. Osmolality was then calculated from biochemical analysis results using each of the 18 formulae. The authors found that the closer an osmole gap is to 0 mOsm/kg, the more accurate the formula and the better it estimates osmotically active solute in the serum. Of the 18 formulae evaluated, the authors recommended the following for calculating osmolality: 2(Na+) + [glucose/18] + [BUN/2.8].

Using this formula, the median osmole gap for the 250 dogs was -2 mOsm/kg. The authors found that this formula was accurate and simple, and it uses readily available serum values.

Commentary

This intriguing study puts to rest a question asked by many criticalists: what is the best formula to use for calculating serum osmolality? Needed to determine the osmole gap, the numbers can depend on what formula you pick. While definitely not an everyday calculation for most clinicians, understanding osmolality and the osmole gap is helpful for diagnosis and management of some toxins, as well as guiding fluid therapy in the ICU. I have long used the formula that the authors recommend. There are easier ones, but the recommended formula takes enough variables into account to be consistently accurate on those rare occasions when it’s needed.—Tony Johnson, DVM, DACVECC

Source


Research Note: Dust, PBDEs, & Feline Hyperthyroidism

Polybrominated diphenyl ethers (PBDEs) are flame retardants found in a variety of consumer products. PBDEs may leach from these products over time and contaminate surfaces and dust in their environment. This contamination produces increased levels of PBDEs in people and animals. Cats have been shown to have 20× to 100× higher levels of PBDEs in their systems than people, possibly through the ingestion of PBDE-contaminated dust when grooming.

This study investigated a possible link between PBDEs and feline hyperthyroidism. The cause of feline hyperthyroidism is currently unknown, but PBDE exposure has been associated with increased thyroid hormone levels and/or decreased thyroid stimulating hormone levels in people. Serum PBDE levels were compared in hyperthyroid cats (n = 35) and age-matched euthyroid cats (n = 30). These levels were also compared to levels found in environmental dust and levels in the local human population. The levels found in cats were higher than those found in the humans, but no difference in PBDE levels or the ratio of different types of PBDEs between hyperthyroid and euthyroid cats was found. There was also no correlation between PBDE levels in feline serum and matched dust samples, although the similar ratios of PBDE types indicates that dust is the likely main exposure route for cats. The authors concluded that exposure to PBDEs is unlikely to cause feline hyperthyroidism, although PBDEs could interact with other factors to influence disease development. The cause of feline hyperthyroidism is likely multifactorial and includes genetic, nutritional, and environmental factors.

Source