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was an early World War II radar system developed by the British Army to provide information for anti-aircraft artillery. There were two upgrades, GL/EF (elevation finder) and GL Mk. II (pictured), both improving the ability to determine a target's bearing and elevation. GL refers to the radar's ability to direct the guns onto a target, known as gun
laying. The first GL sets were developed in 1936 using separate transmitters and receivers mounted on gun carriages. Several were captured in 1940, leading the Germans to believe falsely that British radar was much less advanced than theirs. The GL/EF attachment provided bearing and elevation measurements accurate to about a degree: this
caused the number of rounds needed to destroy an aircraft to fall to 4,100, a tenfold improvement over early-war results. The Mk. II, which was able to directly guide the guns, lowered the rounds-per-kill to 2,750. About 410 Mk. Is and 1,679 Mk. IIs were produced. (Full article...) Recently featured: Andrea Navagero Nosy Komba McDonnell Douglas
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Ngũgĩ wa Thiong'o Kenyan writer and activist Ngũgĩ wa Thiong'o (pictured) dies at the age of 87. In sumo, Ōnosato Daiki is promoted to yokozuna. In association football, Liverpool win the Premier League title. In motor racing, Álex Palou wins the Indianapolis 500. Ongoing: Gaza war M23 campaign Russian invasion of Ukraine timeline Sudanese civil
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(pictured) assumed the office of Thomaskantor in Leipzig, presenting the cantata Die Elenden sollen essen in St. Nicholas Church. 1922 – The Lincoln Memorial in Washington, D.C., featuring a sculpture of the sixteenth U.S. president Abraham Lincoln by Daniel Chester French, opened. 1963 – Buddhist crisis: A protest against pro-Catholic
discrimination was held outside the National Assembly of South Vietnam in Saigon, the first open demonstration against President Ngô Đình Diệm. 2008 – The Convention on Cluster Munitions, prohibiting the use, transfer, and stockpiling of cluster bombs, was adopted. Ma Xifan (d. 947)Colin Blythe (b. 1879)Norris Bradbury (b. 1909)Wynonna Judd
(b. 1964) More anniversaries: May 29 May 30 May 31 Archive By email List of days of the year About Seventeen performing "Oh My!" in 2018 South Korean boy band Seventeen made their debut on May 26, 2015, when they performed a showcase for their debut EP 17 Carat in front of a crowd of 1,000 people. Since then, the group have held 9 concert
tours, 13 fan meetings, and have performed at a number of music festivals and awards shows. Their concert tours include the Right Here World Tour, which sold over one million tickets, and the Follow Tour, which was noted by Billboard as being the top grossing K-pop tour of 2023. In 2024, Seventeen made their first appearances at festivals in
Europe, when they were the first South Korean act to perform at Glastonbury Festival's Pyramid Stage and as headliners for Lollapalooza Berlin. Seventeen's live performances are well regarded by fans and critics alike, and garnered them the award for Top K-pop Touring Artist at the 2024 Billboard Music Awards. (Full list...) Recently featured:
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employee of the Hudson's Bay Company. Tonené was the elected deputy chief before being the lead chief and later the life chief of his community. In his role as deputy, he negotiated with the Canadian federal government and the Ontario provincial government, advocating for his community to receive annual financial support from both. His attempts
to secure land reserves for his community were thwarted by the Ontario premier Oliver Mowat. Tonené's prospecting triggered a 1906 gold rush and the creation of Kerr Addison Mines Ltd., although one of his claims was stolen from him by white Canadian prospectors. This photograph shows Tonené in 1909. Photograph credit: William John Winter;
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hopLength16:48LanguageKoreanLabelPledis EntertainmentLOEN EntertainmentSeventeen chronology 17 Carat(2015) Boys Be(2015) Singles from 17 Carat "Adore U"Released: May 29, 2015 17 Carat is the debut extended play (EP) by South Korean boy group Seventeen. It was released on May 29, 2015, by Pledis Entertainment and distributed by
LOEN Entertainment. "Adore U" serves as the lead single for the EP. 17 Carat features five tracks written, co-written, and co-produced by Seventeen's group members. "Adore U" was chosen as the lead single for the EP and was performed on multiple music shows by the group. "Shining Diamond" was used as a pre-single on the group's reality debut
show. The group stated that the tracklist was chosen to reflect Seventeen's core concept of "boys' passion".[1] The album has two physical versions: one with a "black" themed photo card set, and the other with a "white" themed photo card set. All copies include a CD containing the songs and a fold-up poster/lyric sheet. "Adore U" is the lead single of
the extended play. It was written by Woozi, S.Coups, and Yeon Dong-geon.[2] The Korea Herald states "'Adore U' is a funky pop song about a teenage boy trying to navigate through puppy love."[3] It marks the beginning of the group's trilogy composed of the singles Adore U, Mansae, and Pretty U about a boy meeting, falling in love and asking out a
girl. The track was composed and arranged by Woozi, Bumzu, and Yeon Dong-geon. The music video for the single was released on May 29, 2015, and was directed by Dee Shin. The dance choreography accompaniment to the song was choreographed by Hoshi and focuses on "storytelling, and on highlighting each member's strengths onstage".[4] The
single has sold more than 38,000 digital copies and peaked at number 13 on the Billboard US World Chart. The EP has sold over 82,972 copies in South Korea.[5] It peaked at number 4 on the Korean Gaon Album Chart[6] and number 8 on the US World Billboard Chart.[7] Year-end lists Critic/publication List Rank Ref. Billboard The 10 Best K-pop
Album of 2015 Placed [8] Hoshi participated in the choreography of "Adore U" and "Shining Diamond", Dino choreographed "Jam Jam".[9] Official track list[10]No.TitleLyricsMusicArrangementsLength1."Shining Diamond"WooziVernonS.CoupsKim Min-jeongWooziMasterKeyRishiMasterKeyRishi3:242.""Adore U"" (;
Akkinda)WooziVernonS.CoupsBumzuWooziBumzuYeon Dong-geonWooziBumzuYeon Dong-geon3:073."Ah Yeah" (Hip-Hop unit)S. CoupsVernonWonwooMingyuCream DoughnutRishiCream DoughnutRishi3:294."Jam Jam" (Performance unit + Vernon)WooziHoshiDinoVernonWooziCream DoughnutCream Doughnut3:255."20" (Vocal unit)WooziWooziWon
Yeong-heonWon Yeong-heonDong Ne-hyeong3:23 Weekly chart performance for 17 Carat Chart (2015-2023) Peakposition Japanese Albums (Oricon)[11] 46 South Korean Albums (Gaon)[12] 4 US World Albums (Billboard)[13] 8 Year-end chart performance for 17 Carat Chart (2015) Peakposition South Korean Albums (Gaon)[14] 47 ^ "Seventeen hopes
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page for transcluding these entries Showing 50 items. View (previous 50 | next 50) (20 | 50 | 100 | 250 | 500)Main Page (links | edit) Pledis Entertainment discography (links | edit) List of 2015 albums (links | edit) 2015 in South Korean music (links | edit) Seventeen (South Korean band) (links | edit) S.Coups (links | edit) Vernon (rapper) (links | edit)
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(links | edit) Bumzu (links | edit) Boys Be (EP) (links | edit) You Make My Day (links | edit) You Made My Dawn (links | edit) Jun (Chinese entertainer) (links | edit) List of Stray Kids live performances (links | edit) The8 (links | edit) An Ode (links | edit) Seungkwan (links | edit) Fallin' Flower (links | edit) Heng:garæ (links | edit) Semicolon (EP) (links |
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Right (Seventeen song) (links | edit) 24H (EP) (links | edit) We Make You (links | edit) Hot (Seventeen song) (links | edit) Dream (Seventeen EP) (links | edit) BSS (band) (links | edit) FML (EP) (links | edit) Super (Seventeen song) (links | edit) Always Yours (album) (links | edit) Seventeenth Heaven (links | edit) View (previous 50 | next 50) (20 | 50 | 100 |
250 | 500) Retrieved from " WhatLinksHere/17_Carat" SymptomsCausesDiagnosisComplicationsTreatmentOutlookTakeawayHypervolemia occurs if your body retains too much fluid. You can experience swelling, discomfort, and other symptoms. Untreated, hypervolemia can cause severe complications, including heart failure.Hypervolemia, or fluid
overload, occurs when your body holds onto more fluid than it needs, leading to swelling and other complications.Fluids in the body include:waterbloodlymphatic fluidIf the amount of fluid gets too high, it can impact how it's moved through your body and negatively affect your organ function.Keep reading to learn the signs and causes of hypervolemia
and how doctors diagnose and treat the condition.The symptoms of hypervolemia can include:swelling, also called edema, most often in the feet, ankles, wrists, and facediscomfort in the body, causing cramping, headache, and abdominal bloatinghigh blood pressure caused by excess fluid in the bloodstreamshortness of breath caused by extra fluid
entering your lungs and reducing your ability to breathe normallyheart problems, because excess fluid can speed up or slow your heart rate, harm your heart muscles, and increase the size of your heartincreased weight, caused by excess fluidMedical emergencyIf you experience severe symptoms, such as difficulty breathing, severe pain, or irregular
heart rhythm, call 911 or your local emergency services, or visit a local emergency department.Often, problems with your kidneys cause hypervolemia. This is because the kidneys normally balance the salts and fluids in your body. But when they retain salt, they increase the body's total sodium content, which increases your fluid content.The most
common causes of hypervolemia can include:heart failure, specifically of the right ventriclecirrhosis, often caused by excess alcohol consumption or hepatitiskidney failure, often caused by diabetes and other metabolic disordersnephrotic syndrome, a disorder that causes excess excretion of protein in the urinepremenstrual edema, or swelling that
occurs prior to your menstrual cyclepregnancy, which changes your hormonal balance and can result in fluid retentionYou can also experience hypervolemia from being on an IV, which can cause your sodium levels to be unbalanced. It can also occur if you consume too much sodium.If you believe you're experiencing hypervolemia, speak with a doctor.
They can determine if you're experiencing this condition.First, a doctor typically conducts a physical exam. The key signs of hypervolemia include weight gain and swelling. One or more parts of your body may appear swollen, depending on whether you have been sitting, lying, or standing before your visit.The doctor is also likely to perform a blood test
to check your sodium levels. While your body's total sodium levels will appear elevated if you have hypervolemia, your sodium levels in the blood work may be high, normal, or low.Performing a sodium test on your urine can help determine if your kidneys are causing your hypervolemia or if there is another cause. For renal failure, urinary sodium
content is typically greater than 20 milliequivalents per liter (mEq/L), while in cases of heart failure, cirrhosis, and nephrotic syndrome, it is typically less than 10 mEq/L.If you are hospitalized, your care team may measure your fluid intake and output and your weight to check for hypervolemia. Untreated hypervolemia can cause several complications,
some of which can be life threatening. These can include:pericarditis, or swelling of the heart tissuesheart failuredelayed wound healingtissue breakdowndecreased bowel functionTreatment of hypervolemia differs from person to person, depending on the cause of the condition. Generally, people with hypervolemia may receive a round of diuretics.
These medications remove excess fluid.In severe cases, a doctor may recommend dialysis (fluid removal through the kidneys) and paracentesis (fluid removal through the abdomen).A doctor may also require you to restrict your dietary sodium intake.While you recover from hypervolemia, a doctor may request that you weigh yourself daily to ensure
you're expelling the excess fluid from your body.Many people who stick to a doctor's treatment plans fully recover. This can be important for preventing severe complications.If an underlying condition is causing your hypervolemia, treating the underlying condition may help your recovery.Besides monitoring your weight, you can prevent a recurrence
of fluid overload by:tracking your fluid intakefollowing the fluid intake guidelines from a doctormanaging your thirst with sugar-free candies, ice chips, frozen grapes, and other low-fluid, thirst-quenching foodsensuring you do not consume too much sodiumHypervolemia, or fluid overload, happens when there is too much fluid in your body. It can raise
blood pressure, cause swelling, and impact organ function.Doctors can diagnose and manage hypervolemia with medication, reduced fluid and sodium intake, and dialysis. Many people with this condition can make a full recovery with proper treatment. Symptoms of hypervolemia range in severity for each person and could include:Swelling in an area of your body, most often your arms and legs, where it appears larger than it was a day ago.Bloating in your stomach.Mild discomfort like cramping or a headache.Quick weight gain.Severe symptoms of
hypervolemia that need immediate treatment include:High blood pressure.Shortness of breath.Your heart doesn't pump blood as it should (heart failure).If you have any serious symptoms, visit the emergency room immediately.What causes hypervolemia?Several factors could cause hypervolemia including too much salt in your body, an underlying
condition or hormonal changes.Salt (sodium)Too much salt (sodium) in your body causes hypervolemia. Salt is an essential mineral in your body. Your body manages the amount of salt you eat with water. If you eat food that contains too much sodium, your body will use water to balance it back to a normal level. This is why you might feel thirsty after
eating a lot of salty foods.If you receive intravenous (IV) fluids after surgery or if you're dehydrated, some fluids contain sodium. It's possible to experience symptoms of hypervolemia while getting fluids from an IV because your body's sodium levels aren't balanced.Underlying conditionSome conditions affect how your body manages fluid. Common
conditions that could cause hypervolemia as a symptom include:Heart failure.Kidney conditions.Cirrhosis.Hepatitis.Diabetes.Certain medicines to treat these conditions may cause hypervolemia as a side effect, especially drugs to treat blood pressure or pain management.Treating or managing the underlying condition could resolve hypervolemia and
prevent it from coming back.Hormonal changes and pregnancyChanges to your hormones can cause hypervolemia, most often during your period or during pregnancy when your body retains more sodium and water. Pregnant women often experience swelling in their legs or ankles because the uterus puts pressure on the blood vessels in the body's
lower trunk. This pressure prevents fluid from moving freely through your circulatory system.Why are the kidneys important to regulate fluid in my body?Your kidneys help your body remove excess fluid through your urinary system, which leaves your body as pee (urine). Your kidneys function to balance the amount of fluid in your body, either by
removing it or reusing it. Your kidneys are filters. They separate water and electrolytes (essential minerals including sodium and potassium) from waste. Waste leaves your body and the fluid that's remaining gets recycled into your circulatory system to help your cells and organs function. 2021 Jun 29;8:668688. doi: 10.3389/fvets.2021.668688 Fluid overload (FO) is characterized by hypervolemia, edema, or both. In clinical practice it is usually
suspected when a patient shows evidence of pulmonary edema, peripheral edema, or body cavity effusion. FO may be a consequence of spontaneous disease, or may be a complication of intravenous fluid therapy. Most clinical studies of the association of FO with fluid therapy and risk of harm define it in terms of an increase in body weight of at least
5–10%, or a positive fluid balance of the same magnitude when fluid intake and urine output are measured. Numerous observational clinical studies in humans have demonstrated an association between FO, adverse events, and mortality, as have two retrospective observational studies in dogs and cats. The risk of FO may be minimized by limiting
resuscitation fluid to the smallest amount needed to optimize cardiac output and then limiting maintenance fluid to the amount needed to replace ongoing normal and pathological losses of water and sodium. Keywords: fluid overload, hypervolemia, edema, effusion, fluid balance, resuscitation, stabilization The mammalian stress response to injury,
hypovolemia, or critical illness includes retention of sodium and water and, at least early on, increased thirst (1–5). These responses may serve to defend blood volume and maintain hydration when access to water is impaired by debility, and in the absence of medical care likely confer some survival advantage. However, the application of modern
intensive care sets the stage for harm when potentially limitless amounts water and sodium can be administered to patients whose upset biology favors retention of both. Although evolutionary pressure likely selected for the adaptive responses to hypovolemia following injury or illness, there was no such selection pressure to respond to hypervolemia
in the same setting, a situation now commonly referred to as fluid overload (FO). The concept of FO as a clinical entity to be avoided appeared in earnest within the medical literature during the 1970's, with 54 PubMed citations from that decade using that phrase. Although for many years FO has been recognized as a potential complication of anuria in
chronic hemodialysis patients, in this century the phrase has more often been used to describe a complication of fluid therapy in any patient at risk for hypervolemia or edema. The importance of FO has been underlined by a growing number of reports of observational studies that associate the condition with higher morbidity and mortality in
hospitalized patients, an association that held true in recent meta-analyses of 44 studies in children (6) and 31 in adults (7). Clinically, FO is usually defined by some combination of edema, excessive weight gain, or excessively positive fluid balance in a patient that has received intravenous fluid therapy. In fact, FO is almost universally defined in such
terms in clinical studies of humans (7, 8). However, some have argued that rather than focusing on development of edema as a foundational feature of fluid overload, clinicians should be more concerned about the presence of hypervolemia (9). Hypervolemia is a state of excessive blood volume and elevated mean circulatory filling pressure (MCFP).
Mean circulatory filling pressure is in turn defined as the average transmural pressure of the circulatory system when the heart and blood flow is stopped, and it is determined by blood volume and autonomic control of vascular smooth muscle. Its value is typically close to the average transmural pressure at the level of systemic post-capillary venules,
and the pressure gradient between those venules and the right atrium (central venous pressure) is the driving force returning blood to the heart (10). In animals with a normally functioning heart, intravenous fluid therapy increases cardiac output primarily by increasing the pressure difference between the MCFP and the central venous pressure. If the
MCFP becomes sufficiently elevated by circulatory failure or fluid overload, the elevated venule pressure requires higher capillary pressures to maintain blood flow, and increased capillary pressure promotes fluid movement to the interstitial space. A high rate of fluid administration alone is not enough to produce FO (at least when within a clinically
relevant range); development and maintenance of FO requires impaired excretion of water or abnormal function of the interstitial compartment, or both. For example, administration of 90 mL/kg of lactated Ringer's solution in 1 h to mildly dehydrated dogs produces none of the features of FO, and some features of FO seen during administration of 360
mL/kg in 1 h largely resolve within 30 min, accompanied by voiding large quantities of urine (11). Hypervolemia secondary to impaired excretion of excess fluid may be seen in animals with heart, kidney, or liver disease, and some animals with these chronic conditions present for care precisely because they have clinical signs of FO. Organ dysfunction
as a component of acute illness—for example, impaired heart and kidney function observed in some dogs with septic shock—may contribute to FO in the face of overzealous replacement fluid administration. Another factor contributing to impaired excretion of excess water is dissociation of arginine vasopressin (AVP) release from osmoregulation, a
situation brought about by disorders and drugs that result in water retention, hyponatremia, and edema. As reviewed by Moritz and Ayus, the list of conditions associated with hospital-acquired hyponatremia due to excessive secretion of AVP and water retention is quite long and varied (12). Water retention is a particularly important issue during
treatment of hospitalized children with hypotonic maintenance fluids, who often receive an excess of water via the commonly used Holliday-Seger formula1 (13). Although FO is an occasional complication, neurological consequences of hyponatremia are the more serious side effects of excessive administration of hypotonic fluid to patients prone to
water retention. Stimuli for excessive AVP release during inflammatory states includes an increased plasma concentration of interleukin-6, particularly during sepsis and tissue injury from trauma. Local production of interleukin-6 in response to osmotic stimuli is a physiological stimulus of hypothalamic AVP production, and elevated plasma
concentrations secondary to systemic illness (sepsis in particular) appears to have similar effects on AVP production and release, independent of osmoregulation (14). As anyone who has examined a patient with a soft tissue infection will recognize, inflammation also promotes edema in ways that are independent of volume status and AVP release. As
described by Bhave and Neilson inflammatory states yield a reduction in interstitial fluid pressure by disrupting the tension of the collagen fibrils responsible for maintaining a tight interstitial matrix, increasing the compliance of the interstitial compartment (15). Under physiological conditions, interstitial fluid pressure is under local control via
cellular connections to the collagen matrix through integrin receptors that are in turn linked to the cellular actin cytoskeleton. Inflammation may cause depolymerization of the cytoskeleton and break the integrin links to collagen, loosening the matrix and causing interstitial fluid pressure to fall, favoring fluid movement from the capillary. Regardless
of whether edema was initially caused by an increased MCFP or reduced interstitial pressure, once interstitial fluid volume has grown enough to increase tissue weight by more than 10–20% the compartment becomes highly compliant (16). This increase in compliance allows the interstitial compartment to accommodate large quantities of additional
fluid without much of an increase in pressure, serving to maintain edema once it has begun. In the case of edema caused by intravenous fluids administered to animals with systemic inflammation, there are also contributions from reduced plasma albumin concentration and disruption of the endothelial barrier to albumin. The dilutional effect of fluid
therapy and the acute phase response to reduce plasma albumin concentration decreases plasma oncotic pressure and favors fluid filtration. The capillary glycocalyx barrier to albumin may be compromised by inflammation (17), release of atrial natriuretic peptide secondary to hypervolemia (18), and rapid fluid administration (even in the absence of
increased atrial natriuretic peptide) (19). Thus, both underlying disease and the fluid resuscitation to support the circulation can favor development of edema. FO causes harm due to the effects of edema fluid in the interstitial space. In the lung, the presence of excess extravascular water impairs gas exchange, reduces pulmonary compliance, and
increases the work of breathing, complications that reduce the oxygen content of blood and increase the amount of oxygen consumed by the muscles of ventilation. In the systemic circulation, FO may impair diffusion of oxygen and energy substrates, obstruct capillary blood flow and lymphatic drainage, distort tissue architecture, and impair cell-to-cell
interactions. Every major organ system may manifest complications of the syndrome (Table 1), but the lungs and organs confined by rigid structures (brain) or capsules (kidney, liver) may be particularly vulnerable. It is important to recognize that by the time edema can be seen and felt at the body surface it is also occurring internally. Whereas, the
skin can survive prolonged periods of reduced oxygen delivery and can maintain some of its barrier function despite distorted architecture, internal organs with higher basal oxygen consumption and more complex functional architecture cannot. Adverse effects of FO on organ function. Organ system Potential complications Examples of evidence Brain
•Cognitive impairment •Delirium •Increased ICP/decreased CPP •Mechanically ventilated patients with FO have longer periods of delirium/coma after extubation (20) •Cortical necrosis observed in a dog with FO (21) Gastrointestinal tract •Ileus •Increased permeability to bacterial translocation •Impaired liver function •Increased intra-abdominal
pressure/!!breakcompartment syndrome •Prolonged ileus is associated with FO in humans (22) •Higher wound infection rates associated with FO (23) •Strong association between resuscitation fluid and compartment syndrome (24) Heart •Myocardial edema •Arrhythmia •Impaired systolic and diastolic function •Elevated troponin and pressor
requirements associated with fluid resuscitation (25) Kidneys •Increased interstitial pressure •Decreased RBF/GFR •AKI •Association of FO with AKI (26) Lungs •Pulmonary edema •Pleural effusion •Improved lung function in patients treated with conservative fluid therapy (27) Skin/muscle •Edema •Weakness •Delayed healing of abdominal wound
closure (28) Although FO is often first diagnosed based on recognition of edema or effusion, a better approach is to identify it earlier by monitoring changes in body weight or cumulative fluid balance. Measuring change in body weight has been considered the gold standard clinical measurement approach to monitor fluid balance in hospitalized
humans since the 1970's and has been used to monitor critically ill companion animals (29, 30). However, because of the difficulty in obtaining and charting accurate weights in critically ill humans, a more common strategy is to monitor "ins and outs," that is, cumulative fluid administration vs. the sum of cumulative urine production, drainage losses,
and sometimes volume estimates of diarrhea and insensible losses. This approach requires an indwelling urinary catheter or other means to accurately quantify urine production. Fluid balance is often used as a surrogate for changes in weight, but some studies in adults (31, 32) and neonates (33) have demonstrated extremely poor correlation between
the two measurements. Technical reasons for the discrepancy include charting errors in recording fluid balance and inaccurate/erratic techniques used to obtain multiple weight measurements. One small study of 32 human cardiac surgery patients comparing FO to WG calculations identified 25 arithmetic errors in nursing charts, a finding that
emphasizes the potential for caregiver error to interfere with even objective patient assessments (32). Other potential reasons for discrepancies include failure to accurately measure gastrointestinal losses or loss from wound or body cavity drainage, inaccurate prediction of insensible water losses, and the unpredictable rate of catabolic loss of tissue.
The advent of more widespread use of intensive care beds with built-in scales may make routine use of weight change more practical for human patients. Veterinary application of weight change assessment in critically ill patients is impeded by several factors, including the expense of purchasing and maintaining the calibration of accurate scales, the
need to lift patients onto scales, and the weight effects of monitoring devices, bandages, and bladder size in patients that often weigh less than human infants. Commonly used formulas for % weight gain and % fluid overload include these:
Weight gain (%)=Current (or maximal) body weight - baseline body weightbaseline body weight× 100Fluid overload (%)=Fluid intake - fluid outputbaseline body weight× 100 The data used to populate these formulas may reflect the entire duration of hospitalization or may be used to monitor daily changes. In patients judged to be dehydrated at
baseline, the formula may be modified to subtract the % dehydration (expressed in weight or volume of fluid, depending on the formula) from the numerator (34). Although there is not universal agreement on what degree of weight gain or FO represents a clinically important change, widely cited figures include 5 and 10%, with 10% marking a
threshold for intervention. The development of complications of FO probably depends not only on weight change or fluid balance, but also on the distribution of excess water. In critical illness there may be a variable relationship between total body water content and the compartmental distribution of water between extravascular and intravascular, and
within the vascular compartment the distribution between the unstressed volume and the stressed volume that creates cardiac preload. An excess of extracellular water is more likely to produce clinical complications of FO than an excess of intracellular water. Supporting this is the observation of Bihari et al. that sodium balance—a key determinate of
extracellular fluid volume—correlates better with respiratory dysfunction than does fluid balance (35). The presence of hypervolemia has been assessed by physical exam findings, invasive hemodynamic pressure measurements, and ultrasound assessment. As reviewed by Beaubien-Souligny et al. (36), ultrasound techniques may identify hypervolemia
in humans by documenting internal jugular vein distension, dilation of the inferior vena cava, reversal of systolic:diastolic hepatic vein flow, development of pulsatile portal vein flow, and discontinuous intrarenal venous flow. Ultrasound manifestations of increased intracranial pressure include an increase in the diameter of the optic nerve and a
reduction in diastolic flow of the middle cerebral artery. The presence of increased extravascular lung water is revealed by an increase in the number of B lines. Ultrasound examination is also used to identify patients that are likely to respond to intravenous fluids with an increase in cardiac output. Veterinary studies using ultrasound to predict fluid
responsiveness have included assessment of caudal vena cava collapsability (37, 38) and prospective comparison of the CVC diameter to that of the abdominal aorta (38), where "fluid responsiveness" was defined as a >15% increase in ascending aorta velocity time index immediately following administration of a fluid bolus. Although the authors of a
retrospective case series (37) concluded that the effect of administration of 30 mL/kg of LRS (at an unspecified rate) could be predicted by the magnitude of the respiratory effect on CVC diameter, a prospective study demonstrated that respiratory variation in CVC diameter did not predict the response to rapid (1 min) administration of 4 mL/kg of
Hartmann's solution. In contrast, a ratio of the maximum CVC diameter to aortic diameter at the level of the porta hepatis of 0.83 had a sensitivity of 100% and a specificity of 75% for fluid responsiveness. The author is aware of just three reports of small studies that addressed the incidence and impact of FO in dogs and cats. A retrospective study
defined FO as development of symptomatic pulmonary edema or pleural effusion in 11 cats with urethral obstruction that were treated with intravenous fluids, and compared recorded data from those cats with 51 control cats that had urethral obstruction but did not develop respiratory signs (39). A "FO score" was calculated based on the % fluid
overload formula (see above), and weight change between admission and the date that respiratory signs developed was calculated. Although the relationship between FO score and weight change was not described, some cats in both groups had negative fluid balance and some affected cats lost weight by the time they developed respiratory signs.
Although the range of FO scores in the control cats was greater than that of the cats with FO, and included subjects with more severely negative and positive fluid balance, the median FO score in affected cats was significantly greater than the controls (6 vs. 2.46%). Ten affected cats developed a cardiac gallop, and echocardiography identified
underlying heart disease in 5 of 6 cats examined; therefore, occult heart disease was likely the single most important factor in the development of clinical signs. Another veterinary report defined FO as a positive fluid balance in dogs (after correcting dehydration) that were monitored with a closed urine collection system, and compared outcomes
between 34 dogs with critical illness and 15 hemodynamically stable dogs with neuro-orthopedic disease that had closed urine collection systems in place to assist with nursing care (34). Fluid balance and % FO were examined as continuous data, and correlation with APPLE scores and survival at discharge was evaluated. Critically ill dogs had
significantly greater positive fluid balance than the control group, and 8/16 dogs with substantial FO (12% or more) died. There was only a weak correlation between % FO and composite APPLE scores, which were based on clinical data that was not collected at a standardized time point and could have under- or overestimated the severity of illness.
More recently, a prospective observational study of dogs with acute kidney injury described the relationship between systemic hypertension and severity of kidney injury, and included observations about FO (40). In this report, FO was characterized as edema and was diagnosed based only on discretionary clinician assessment of acute weight gain,
development of body cavity effusion, or physical examination findings. 22/52 dogs met the criteria for FO, and these dogs were significantly more likely to have hypertension and were more likely to die than dogs without. Although these studies demonstrate potential harm of FO in clinical veterinary patients, a causal relationship between FO and
outcome can't be demonstrated by a retrospective or observational study. Prospective interventional studies comparing the effect of standard (or liberal) fluid administration with fluid restriction on %FO and outcomes are needed to address this. Most reports of studies of FO have focused on the effect of fluid administration in the early hours to 1–2
days of treatment of life-threatening illness. Because of the growing evidence that FO is associated with worse outcomes in the critically ill, there has been considerable interest in validating non-invasive techniques to identify patients who respond to intravenous resuscitation fluid with an increase in cardiac output, and avoid (or at least limit)
administration of fluids to those who do not. Those techniques are reviewed in detail by Boysen and Gommeren elsewhere in this issue; however, it is worth mentioning here that there are unresolved questions about the benefit of an increase in cardiac output immediately following a fluid bolus when that increase may be transient and yet result in
longer-lasting edema. For example, Roger et al. demonstrated that of the septic patients who responded to a fluid bolus with an increase in cardiac stroke volume, half lost that benefit within 20 min (41). Most studies of techniques used to predict fluid responsiveness have not characterized patient responses beyond a few minutes, and it is quite
possible that for many, a transient response to fluid infusion does not translate into a sustained benefit for circulation or outcome. Fluid therapy does not end with initial resuscitation and in fact often continues for days in the critically ill, in stages that have been characterized as rescue, optimization, stabilization, and de-escalation (Figure 1) (42).
During initial rescue (resuscitation), most clinical decisions about fluid therapy are made within minutes and are based predominately on clinical signs. It is common to see unambiguous signs of positive hemodynamic responses to fluid administration in overtly hypovolemic patients, and physical examination alone or sometimes in combination with the
ultrasound techniques noted above is often adequate to guide fluid dose and rate of administration and avoid hypervolemia. The optimization phase occurs over hours, or longer if the underlying disease is complex or progressive, for example sepsis or pancreatitis. During this phase fluid therapy may require ongoing administration of replacement type
fluids that are titrated toward optimizing the circulation but avoiding an excess that will produce edema. It is at this stage that response to fluids may become much more nuanced and difficult to evaluate with physical examination alone, and using other techniques—for example, ultrasound and measurements related to oxygen delivery—become much
more important. The stabilization phase occurs during recovery when the patient has become hemodynamically stable and fluid therapy shifts toward optimizing electrolyte balance, replacing normal and pathological ongoing losses, and the beginning of a negative fluid balance as the patient excretes the excess fluid administered during resuscitation
and optimization. The de-escalation phase is characterized by a transition to self-sufficiency via oral intake and a negative fluid balance where FO had occurred. Conceptual relationship between patient volume status and the four phases of fluid therapy in critical illness. Open access image from the Acute Dialysis Quality Initiative 12, downloaded from
on 3/15/2021.The optimization and stabilization phases are times when development of FO due to inappropriate administration of excessive quantities of both water and sodium in "maintenance" fluids and fluid vehicles for drug administration is probably common. Routine practice in charting human fluid balance ignores the volume of fluid
administered as a vehicle for drugs, and this "fluid creep" can create a large volume of unaccounted fluid administration that contributes to FO. In fact, maintenance fluid and fluid creep may account for the majority of parenteral water and sodium administration in human ICU patients (43). The common veterinary practice of using a high-sodium
replacement fluid like lactated Ringer's or a proprietary equivalent, at volumes that are often much larger than required to replace normal ongoing losses in an immobilized critically ill dog or cat, likely contribute a great deal to the prevalence of FO. Maintenance requirements of water and electrolytes for dogs and cats may be very different from
commercial fluid composition and commonly cited administration rates, as has been recently reviewed in detail elsewhere (44). The clearest indication for intervention in animals with FO is the presence of hypervolemia, which should be managed with sodium and water restriction, diuretics, and in selected life-threatening situations, hemofiltration
when the potential for a diuretic response is impaired. The most obvious cases of hypervolemia include edematous animals with kidney injury causing oliguria or anuria, and animals with pulmonary edema secondary to heart failure. Physical examination alone is likely to correctly identify these and inform treatment. Diuretic treatment for left sided
congestive heart failure usually consists of furosemide 1–2 mg/kg by intravenous or intramuscular injection, followed by an intravenous constant infusion (0.66 mg/kg/hour) for 6 h treatment blocks when venous catheterization can be obtained without compromising the patient (45). Animals with kidney injury and oliguria may have impaired delivery of
the drug to site of action in the loop of Henle. Animals that do not respond to usual doses of furosemide (e.g., 2 mg/kg) may respond to high doses, e.g., 2 mg/kg repeated to as much as 8 mg/kg within 1–2 h, followed by a continuous infusion titrated to maintain the target rate of urine production. Animals with other causes of FO and edema may be
hyper-, normo-, or hypovolemic, and classification and treatment of these may be more difficult. For example, an animal with sepsis or pancreatitis may become edematous at a low MCFP because of changes in the microcirculation and interstitial matrix, and aggressive treatment with diuretics for imagined hypervolemia will compromise circulation and
cause harm. These animals may develop relatively severe FO after even tiny increases in MCFP. If there is clinical evidence of impaired circulation that may respond to fluid therapy, benefit from resuscitation fluids should be predicted and monitored with adjunct assessment methods such as ultrasound measurement of dynamic variables, central or
mixed venous oxygen saturation, and central venous or arterial blood pressure responses to incremental doses of fluids or drugs. Replacement fluids should be used only during the resuscitation and optimization phases, with an eye toward discontinuing them as soon as a transition to the stabilization phase is possible. Albumin solutions or plasma may
be administered to animals with hypoalbuminemia that is sufficiently severe to contribute to edema formation. Although administration of albumin solutions instead of crystalloids to critically ill humans provides little improvement in most outcome measures, those patients generally require less total fluid for resuscitation/optimization and may have
less tendency for FO (46–48). We have observed similar benefit from albumin or plasma administration to critically ill dogs in our ICU, reaching the stabilization phase with less tendency for FO. Although fresh frozen plasma is an inefficient way to provide albumin, some animals may benefit from coagulation factors or other plasma components. In our
ICU, a common dosing strategy for animals in shock from sepsis and other causes of a systemic inflammatory response is to use plasma (and more recently, 5% canine albumin) as a component of resuscitation fluid therapy, then continue administration as a continuous rate infusion of ~20 mL/kg/day during the optimization phase. This fluid is always
accounted for in total fluid balance calculations, and crystalloid administration is reduced by an equal amount. Animals in septic shock are rarely resuscitated with fluid therapy alone. Removal of the underlying cause, for example infection source control, is a critical step to reversing pathology of the microcirculation and interstitial matrix to correct
FO. Mechanical ventilation to reduce the work of breathing and decrease pulmonary shunt may be required to address an increase in extravascular lung water. Pharmacological management of the circulation, for example the administration of pressors to maintain MCFP, arterial pressure, and cardiac output, is routinely essential to correct hypotension
restore adequate oxygen delivery and allow earlier transition to a stabilization phase with less fluid administration. Once an animal with FO edema has reached the stabilization phase it may be much more tolerant of fluid restriction and graded diuretic therapy. In this stage, a low test dose (