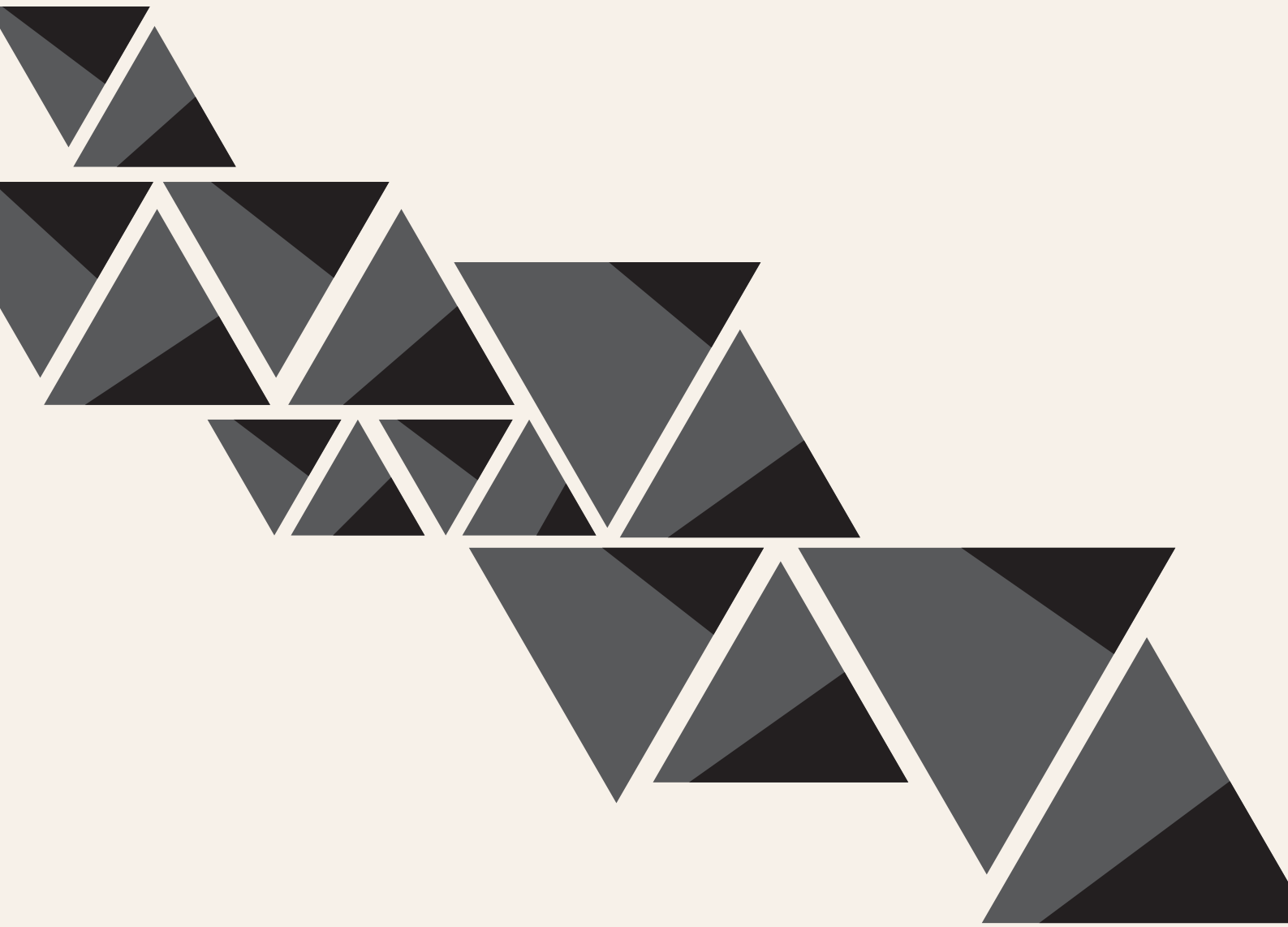
January-March 2016

ISSN 2222-5188

Volume 13 Issue 1

info MEDICUS

The essence of medical practice



Maintenance of Intravenous Fluid

Contents

Published by

Medical Services Department
ACI Limited
Novo Tower, 9th Floor
270 Tejgaon Industrial Area
Dhaka-1208

Designed by

Creative Communication Ltd.
Road # 123, House # 18A
Gulshan 1, Dhaka 1212

Editorial

Dear Doctor,

Happy New Year 2016!

On behalf of ACI Limited, we wish you a very healthy and prosperous life throughout this New Year and beyond.

Reflecting on our topics in this issue, to begin with, the first line treatment for acutely ill patient is intravenous fluid. The goal of intravenous fluid is to preserve the extracellular volume while maintaining a normal electrolyte balance. Maintenance of intravenous fluid of an ill patient is a great change for a physician; that's why in the Review Article section; we have discussed various aspects of "Maintenance of intravenous fluid".

There are many serious conditions where long term vascular access is necessary in neonate for the delivery of medication and nutrition. The peripherally inserted central catheter (PICC) provides central vascular access in neonates. For this reason, in the Clinical Method we have portrayed "PICC placement in the neonate".

Medical science is improving rapidly. Many revolutionary inventions are happening in the medical sector. In Health News section we have collected some interesting latest medical information.

In recent times, people are suffering from many neurological disorders worldwide. Among them Bell's palsy is most common which mainly occurs with any problem in facial nerve. "Bell's palsy" is the topic of our View Point.

In addition, in the Clinical Image section we place some clinical pictures and their short description. Hope these would act as valuable information.

Thanks and best regards,

(Dr. S. M. Saidur Rahman)
Medical Services Manager

(Dr. Rumana Dowla)
Manager, Medical Information & Research

Maintenance of intravenous fluid

A critical aspect of the care of patients is the administration of intravenous fluids. Intravenous fluids may be required as a bolus infusion for resuscitation or as a continuous infusion when sufficient fluids cannot be ingested orally. The goal of maintenance intravenous fluids is to preserve the extracellular volume while maintaining a normal electrolyte balance. An appropriate maintenance fluid provides an adequate quantity of both water and electrolytes to ensure good tissue perfusion without causing complications related to fluid overload or volume depletion. It also prevents the development of hyponatremia, hypernatremia, and other electrolyte imbalances. Despite the almost everywhere need for intravenous fluids there has been little consensus on the most appropriate rate of administration and composition of intravenous fluids, and practice patterns with respect to maintenance fluids vary widely. In addition, patients frequently have conditions that impair normal water and electrolyte homeostasis and choosing the appropriate volume and composition of intravenous fluids requires great care. Intravenous fluids can be classified, according to the concentration of sodium plus potassium in the fluid, as being either isotonic or hypotonic. The dextrose content of intravenous fluids has no effect on the tonicity, since the dextrose is rapidly metabolized when it enters the bloodstream and should not produce hyperglycemia. However, in this review article we have discussed various aspects of intravenous fluids management.

Mechanism of fluid balance

The human body has a remarkable ability to maintain a normal composition of body water and plasma osmolality, despite wide variations in fluid and electrolyte intake. Sodium and water homeostasis is regulated through the actions of AVP (arginine vasopressin), the renin angiotensin aldosterone system, and natriuretic peptides. Plasma osmolality is regulated both by thirst and by excretion of free water. In a patient who can ingest nothing by mouth, plasma osmolality is primarily under the control of AVP release, which determines the rate of free water excretion. Any disease state that results in either excess AVP or impaired AVP action will place a patient at risk for a plasma sodium concentration that is too high or too low.

There are numerous hemodynamic and nonhemodynamic stimuli for AVP secretion thus, virtually all acutely ill hospitalized patients are at risk for hyponatremia (Figure 1). Hemodynamic stimuli for AVP production include volume depletion, hypotension, edematous states such as congestive heart failure, cirrhosis, and the nephritic syndrome and sepsis. Nonhemodynamic physiological stimuli for AVP secretion include pain, stress, nausea, vomiting, hypoxemia, hypercapnia, hypoglycemia, and the perioperative state.

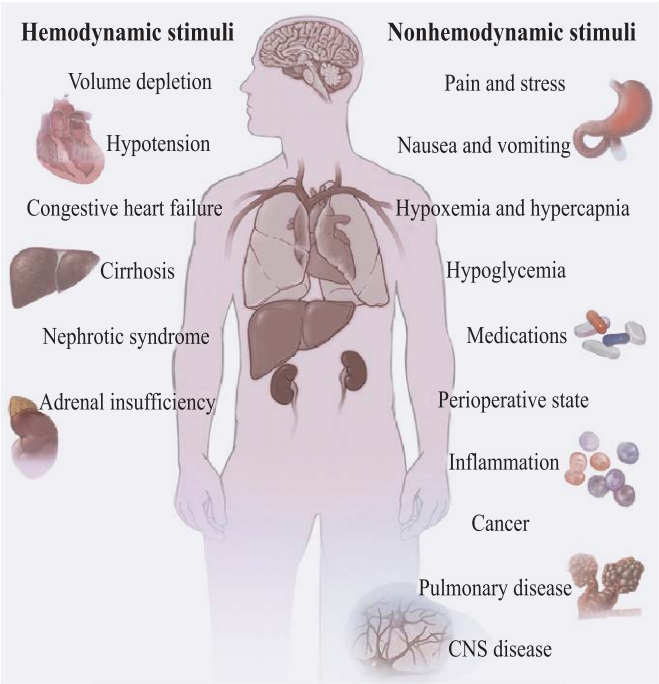


Figure 1: Nonosmotic states of arginine vasopressin (AVP) excess

These physiological stimuli can result in increased AVP levels in the absence of volume depletion or hyperosmolality. In addition, an ever expanding list of conditions and medications is associated with the syndrome of inappropriate antidiuresis, in which AVP excess occurs in the absence of any identifiable nonosmotic stimuli for AVP production. The conditions most often associated with this syndrome are cancer, central nervous system (CNS) disorders, pulmonary disorders, and infections. Medications that are frequently associated with this syndrome include narcotics, the chemotherapeutic agents cyclophosphamide and vincristine, selective serotoninre uptake inhibitors, the antiepileptic agent oxcarbazepine, and the recreational drug "ecstasy" (3,4-methylenedioxymethamphetamine [MDMA]). Thiazide diuretics can also be associated with a condition that resembles the syndrome of inappropriate antidiuresis. The syndrome of inappropriate antidiuresis is now recognized as the most common cause of euvolemic hyponatremia. There are many conditions where intravenous fluid therapy is essential. Some conditions are given in table 1.

Hospital acquired hyponatremia

Hyponatremia, which is defined as a plasma sodium concentration of less than 135 mmol per liter, is the most common electrolyte abnormality in hospitalized patients; it affects approximately 15 to 30% of children and adults who are hospitalized. Most hyponatremia in these patients is hospital acquired and is related to the administration of hypotonic intravenous fluids in patients with elevated AVP levels.

Patients with hospital acquired hyponatremia are at particular risk for the development of hyponatremic encephalopathy, which usually develops acutely, in less than 48 hours, leaving little time for brain adaptation. Hospitalized patients who are at particular risk for the development of hyponatremic encephalopathy at even mildly hyponatremic levels include children younger than 16 years of age, women in their reproductive years, and patients with hypoxemia or underlying CNS disease, since these conditions either impair regulation of brain cell volume or are associated with decreased intracranial capacity for brain expansion.

Table 1. Conditions required fluid therapy

1. Free water restriction for euvolemic states of AVP excess

CNS disturbances

- Meningitis
- Encephalitis
- Brain tumors
- Head injury
- Subarachnoid hemorrhage

Pulmonary disease

- Pneumonia
- Asthma
- Bronchiolitis
- Tuberculosis

Cancer

Postoperative state

2. Fluid restriction for edematous states

- Congestive heart failure
- Nephrosis
- Cirrhosis

3. Fluid and sodium restriction for oliguric states

- Acute glomerulonephritis
- Acute tubular necrosis
- End stage renal disease

4. Increased free water requirements for renal concentrating defects

- Congenital nephrogenic diabetes insipidus
- Sickle cell disease
- Obstructive uropathy
- Reflux nephropathy
- Renal dysplasia
- Nephronophthisis
- Tubulointerstitial nephritis
- Use of lithium

5. Increased sodium and water requirements for solute diuresis

- Diuretic phase of acute tubular necrosis
- Postobstructive diuresis
- Immediate postoperative renal transplantation
- Diabetic ketoacidosis
- Bartter's syndrome
- Fanconi's syndrome
- Adrenal insufficiency

6. Increased free water requirements for extra renal free water losses

- Burns
- Prematurity in neonates
- Fever
- Infectious diarrhea

Prevention of hospital acquired hyponatremic encephalopathy is critical, since the presenting symptoms are nonspecific and can be easily overlooked until advanced symptoms develop. The most consistent symptoms of hyponatremic encephalopathy are headache, nausea, vomiting, and generalized weakness. Advanced symptoms of hyponatremic encephalopathy include seizures, respiratory arrest, noncardiogenic pulmonary edema, and decorticate posturing. Symptoms can occur abruptly and do not always correlate with the plasma sodium concentration or the rapidity of development of hyponatremia.

Nacl, 0.2% Nacl, 2.5% Dextrose water. These fluids are indicated for heart related disorders, fresh water drowning, peritonitis, hypovolumic and hemorrhagic shock, diabetic disorders etc. Parenteral fluid should be given via volumetric pump if a patient is on fluid for over 6 hours or if the fluid contains potassium. Table 2 show the measurement of required fluid according to body weight.

Indications for fluid and sodium restriction

The administration of maintenance fluids at the rate of 1500 ml/m^2/day with 0.9% NaCl is not appropriate for all disease states, and in some disorders it can result in serious fluid overload.

Table 2: Requirement of fluid according to body weight

Weight (kg)	Fluid requirement (ml/24 hours)	Rate (ml/hour)
35 to 44	1200 (10 hourly)	50
45 to 54	1500 (8 hourly)	65
55 to 64	1800 (7 hourly)	75
65 to 74	2100 (6 hourly)	85
≥ 75	2400 (5 hourly)	100 (max)

Failure to recognize and treat hyponatremic encephalopathy with hypertonic saline results in a poor neurologic prognosis. Fluid restriction alone, isotonic fluids, and vaptans have no role in the immediate management of hyponatremic encephalopathy.

Types of fluid requirement

Isotonic solutions

Isotonic solutions have a concentration of dissolved particles equal to that of intracellular fluid. It has osmolality of 250 - 375 mOsm/L. For example, 0.9% Nacl, Lactated Ringer, Ringers' Solution, and 5% Dextrose in water. These fluids are indicated for shock, resuscitation, fluid challenges, blood transfusions, metabolic alkalosis, burns, hypo and hypernatremia.

Hypotonic solution

Hypotonic solutions have fewer particles than does intracellular fluid. Fluid flows into cells. It has osmolality of > 375 mOsm/L or higher. For example, 3% Nacl, 5% Nacl, 3% Nacl or 5% Nacl + Distilled water. These fluids are indicated for hypertonic dehydration, gastric fluid loss, slow rehydration etc.

Hypertonic solution

Hypertonic solutions have a greater concentration of dissolved particles than does intracellular fluid. Fluid is pulled from cells. It has osmolality of > 250 mOsm/L. For example 0.45% Nacl, 0.33%

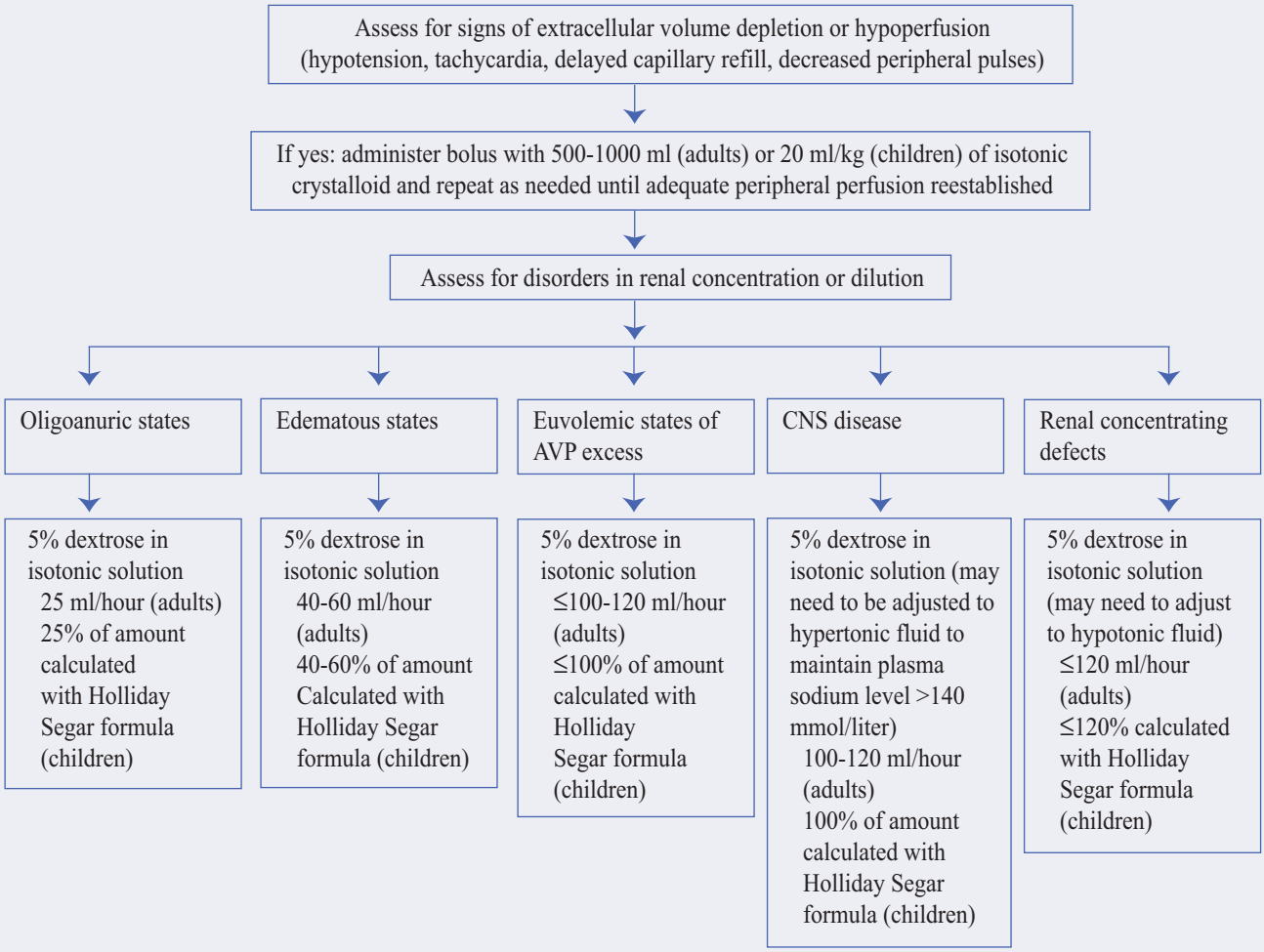
There are certain disease states where the ability to excrete both salt and water is severely impaired and both fluid and sodium restriction is necessary to prevent fluid overload. Disease states where both sodium and water restriction is indicated can be broadly classified as either edematous states or oliguric states. The most common conditions that lead to edema are congestive heart failure, hepatic cirrhosis, and nephritic syndrome. The mechanism of edema formation and its treatment are different in each of these conditions. What they all have in common is an impaired ability to excrete free water due to decreased effective circulating volume, which makes hyponatremia a common associated complication. Oliguric acute kidney injury can result from decreased renal perfusion; ischemic, toxic, or obstructive insults or glomerular or tubulointerstitial inflammatory processes. In prerenal states of acute kidney injury, such as sepsis or hypovolemic shock, fluid resuscitation is essential and life saving.

Guideline of intravenous fluid therapy

No single rate of administration or composition of maintenance intravenous fluids is appropriate in all circumstances. Thus, intravenous fluids may be viewed as medications that require careful dose adjustment that is specific to the disease state of each patient.

The rate of administration and the composition of intravenous fluids need to be individualized, and while patients are receiving intravenous fluids, they require close monitoring with daily measurement of weight, frequent assessment of vital signs, strict measurements of intake and output, and daily measurement of serum electrolyte levels and physical examination. Many children have died of hyponatremic encephalopathy within 24 hours after the initiation of hypotonic intravenous fluids, so even close observation may be inadequate to prevent this complication. Table 3 shows a practical approach for adjusting maintenance intravenous fluids according to these conditions.

Table 3: Guide to maintenance intravenous fluid therapy



Conclusion

The administration of intravenous fluids is an essential component of supportive care for acutely ill patients. Because of limited evidence, recommendations regarding fluid have historically been opinion based. It has now become clear that the administration of hypotonic maintenance fluids (sodium concentration, <130 mmol per liter) is associated with the development of hospital acquired hyponatremia as well as with deaths or permanent neurologic impairment from hyponatremic encephalopathy. Acutely ill patients have multiple stimuli for AVP that place them at risk for the development of hyponatremia. Numerous prospective studies involving children have shown that isotonic fluids are safe and effective in preventing hospital acquired hyponatremia. Acutely ill patients can have a variety of conditions that can alter body water homeostasis, so both the rate and the composition of intravenous fluid should be prescribed carefully. Isotonic fluids are the most appropriate maintenance fluid in the vast majority of situations. However, a solid evidence base from which consensus guidelines for fluid therapy could be built is also needed to better standardize fluid management.

References: *1. N. Eng. J. Med. 1 October, 2015; Vol. 373, N (14)*
2. Curr. Opin. Pediatr. Vol. 186-193, N (23)
3. Fluid prescription working group, 3rd edition, January 2014

PICC placement in the neonate

Neonatal patients often require long term vascular access for the delivery of life sustaining medications and nutrition. The peripherally inserted central catheter (PICC) offers several advantages over other devices used to provide central vascular access in neonates. A neonatal PICC can be inserted with the use of an analgesic agent and radiographic verification, and it can remain in place for several weeks or months. The small diameter of lumen is ideal for the extremely small neonate. The neonatal PICC has a tip that terminates at or close to the heart or in one of the great vessels, the superior vena cava or the inferior vena cava.

Indications

Early identification of patients who require long term vascular access and early central catheter placement decreases the number of skin punctures and increases the success rate of neonatal PICC placement. Candidates for a neonatal PICC include infants with very low birth weight who require continuous parenteral nutrition, infants who require long term intravenous access, infants who require the infusion of fluids or medications that have hyper osmolar or irritating properties, infants who need a dedicated catheter to deliver a critical infusion of life sustaining medication, and infants in whom peripheral access cannot be achieved or maintained.

Vein selection

Commonly used insertion sites are the arms, legs, and scalp. The veins of the hands and feet are other possible sites for neonatal PICC. The basilic vein is larger and less tortuous than the cephalic vein. The right basilic vein is preferred over the left basilic vein, because the right vein has a more direct route to central circulation. The axillary vein is large and provides a direct route to the subclavian vein. Be careful to avoid cannulation of the axillary artery. Scalp veins are easily visualized but may become tortuous at the level of the ear and on entrance to the subclavian veins, moving the patient's head to a midline position during insertion may facilitate catheter advancement to the central circulation. Leg veins offer multiple sites for neonatal PICC placement, but advancing the catheter at the level of the femoral fold can be difficult. Femoral veins and jugular veins are best cannulated with ultrasound guidance.

Equipment

Select the appropriate type of catheter for the patient. Have radiopaque contrast material available if its use is anticipated. Put on a cap and mask before opening sterile equipment. The following equipments are necessary; introducer, measuring tape, antiseptic solution, iris forceps, syringes, gauze, sterile drapes, tourniquet, scissors, trimming device, adhesive skin closure strips, clear occlusive dressing, flush and labels.

Patient preparation

Determine the location for insertion of the catheter, measure the anticipated length of the catheter, and note the length in centimeters. For insertions in the arm or scalp, measure from the insertion site along the course of the vein to the right sternal boarder and to the level of the nipple line or third intercostals space.

For insertions into the leg, measure from the insertion site along the course of the vein to the level of the diaphragm. When using an insertion site in the arm, turn the patient's head toward that arm to help reduce the likelihood that the catheter will be malpositioned in the internal jugular vein.

Catheter insertion

Begin the placement procedure by confirming the patient's identity, and then determine the location for placement. Consider removing oral gastric and nasal gastric tubes, their removal may aid in the visualization of the neonatal PICC on radiography. Neonatal PICC placement is a sterile procedure, and strict precautions should be

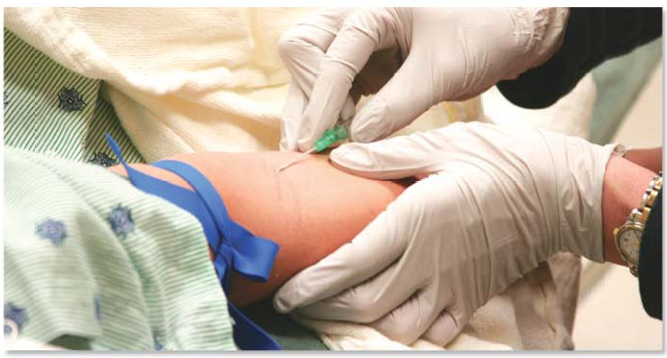


Figure 1: Puncturing of a vein with an introducer needle

observed to preserve the sterile barrier. Prepare the desired insertion site with antiseptic solution and allow it to dry. The choice of antiseptic solution may vary according to institution and patient population. If using povidone iodine, remove it from the patient's skin after it has dried to prevent tissue damage, absorption, and thyroid suppression. It is also important to flush all lumens of the catheter with a saline based solution to ensure that there are no imperfections or breaks in the catheter. When using a catheter with a stylet, never trim the stylet. Pull the stylet back such that its tip is retracted by approximately half a centimeter from the catheter tip before trimming the catheter. Peel away sheaths and break away needles are common introducer types. Inspect the introducer for imperfections.

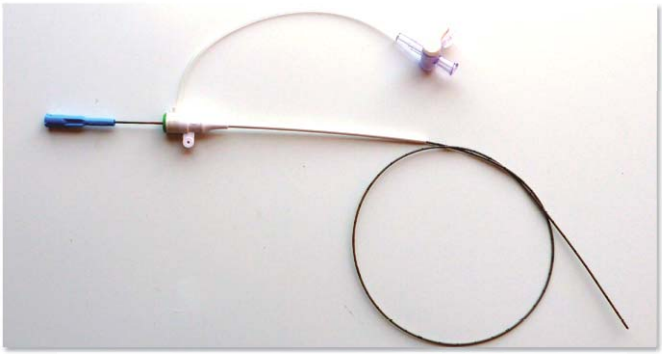


Figure 2: Insertion of a catheter through an introducer

Puncture the vein with the introducer needle (Figure 1) and observe for blood return. Stop advancing the needle as soon as there is blood return to prevent puncture of the posterior wall of the vein. When using a peel away sheath, withdraw the needle and then insert the catheter through the introducer to the appropriate premeasured location (Figure 2). The catheter should advance with ease. Once the catheter is properly located in the inferior vena cava or superior vena cava, blood should return with ease. When using a stylet, ensure that the catheter has been properly flushed and remove the stylet slowly to prevent damage to the catheter. Remove the introducer from the catheter.

Confirmation of catheter placement

Stabilize the catheter with adhesive skin closure strips and obtain a radiograph to confirm the position of the catheter. The ideal location for the catheter tip is parallel to the vessel wall in the central venous system in the superior vena cava or inferior vena cava, just proximal to the right atrial junction. This location is described as being 1 cm outside the heart in a premature infant and 2 cm outside the heart in full term infant. Radiopaque water soluble contrast material may be needed to enhance the visibility of neonatal PICC, at some institutions its use is routine. Administer enough contrast material to approximate the intraluminal volume. The lumens of most neonatal PICC accommodate a volume of 0.3 ml or less.

When the catheter tip lies within the area of the inferior vena cava but does not advance to the region of T8 to T10, or when the catheter takes an unexpected course or bends, kinks, or curls in the area of L2 to S1, it may be malpositioned. Consider obtaining a cross table, lateral film to ensure that the catheter has not entered lumbar or renal venous circulation. The catheter can be erroneously diverted into the ascending lumbar veins before it reaches the inferior vena cava. The ascending lumbar veins arise from the iliac veins at the level of L5 to S1. Renal veins exit at the level of L2. If anyone unable to advance the catheter tip to central venous circulation, they may consider using the catheter as a midline. Position the midline catheter tip so that it terminates in the proximal portion of the leg, parallel to the femur, or in the arm, parallel to the humerus. If the PICC has been placed in the scalp, make sure that the tip terminates in the external jugular vein.

Securing the Catheter

After correct placement has been verified radiographically, stabilize the catheter with adhesive skin closure strips and cover the area with a sterile, clear, occlusive dressing. Maintain visibility of the insertion site, coiling any unused portion of the catheter away from the insertion site. If the catheter has been inserted in an arm or leg, do not encircle the limb with the dressing. Ideal locations for the catheter tip are in the central venous system in the superior or inferior vena cava, just proximal to the right atrial junction and parallel to the vessel wall.

Catheter removal

Central catheters should be removed as soon as they are no longer needed, before removing a catheter, perform hand hygiene. Neonatal PICC should be easy to remove. Cleanse the removal site with the appropriate antiseptic and gently pull out the catheter. Document the length of the catheter that was removed, and check the patient's record to verify that the catheter has been removed in its entirety. When the removal of a neonatal PICC is difficult, it may be necessary to redress the catheter, to flush the catheter, and to then make another attempt at removal after a period of several hours or to obtain a surgical consultation. Do not force catheter removal, because excess tension may fracture it. One technique that may facilitate removal is the application of a warm compress to the tract of the vein for 20 to 30 minutes, flushing of the catheter, and massaging of the area near the insertion site.

Contraindications

In general, there are few contraindications for neonatal PICC placement. Central catheters are often necessary as a "lifeline" for the patient. However, there are certain circumstances in which a central catheter should be avoided if possible. These include situations in which there is an active bloodstream infection (i.e., a positive blood culture within the preceding 48 hours), there is a thrombus in the targeted vein, the parents do not provide consent, and vascular access can be achieved with the use of a peripheral or midline catheter.

Summary

Vascular access is an important component of the care of the sick or preterm neonate, providing life sustaining parenteral nutrition and medications. Neonates should be treated by skilled person who has excellent knowledge of neonatal PICC placement and catheter care.

Reference: *N. Eng. J. Med. 13 March, 2014; Vol. 370, N (11)*

A pregnant woman with acute cardiorespiratory failure: dengue myocarditis

A 29 years old woman, 31 weeks into her fourth pregnancy, presented with a 2 day history of fever, dry cough, chest pain, and shortness of breath. She had a history of partial thyroidectomy for hyperthyroidism in two years back, and had three miscarriages. She took 150 µg thyroxine daily and was a non smoker. She was initially treated for presumed pneumonia with imipenem. However, her respiratory function deteriorated overnight and she was transferred to the intensive care unit (ICU).

On admission, she was febrile (38°C), tachycardic (pulse 115 beats per min), and tachypnoeic (respiratory rate 28 breaths per min), with an SpO_2 of 94% on 5 L oxygen. Heart sounds were normal but she had bibasal crackles on chest auscultation. Apart from a gravid uterus, abdominal examination was normal. Chest radiograph showed bilateral infiltrates suggesting pulmonary oedema. Haemoglobin was 92 g/L, haematocrit 29·2%, total white cell count $8·5 \times 10^9$/L, (neutrophil count $7·53 \times 10^9$/L and lymphocyte count $0·4 \times 10^2$/L), with a platelet count of 134×10^9/L; INR was 1. Urea, electrolytes, liver transaminases, and procalcitonin were normal. Arterial blood gas analysis showed pH 7·49, PaO_2 69 mm Hg, $PaCO_2$ 30·9 mm Hg, HCO_3 23·7 mmol/L, and lactate 1·2 mmol/L. She was giving oseltamivir and continued imipenem and maintenance fluids.

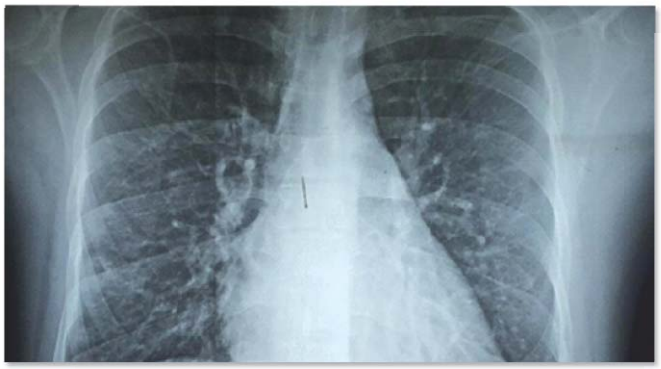


Figure: Dengue myocarditis

Her respiratory function deteriorated further (respiratory rate 35 breaths per min, SpO_2 85% on 15 L oxygen) so doctors started non invasive ventilation with bilevel positive airway pressure (BiPAP) and added vancomycin and azithromycin. ECG showed inverted T waves in the inferior leads and troponin I was raised (1·1 µg/L; normal <0·3 µg/L) peaking at 1·6 µg/L the next day, with pro brain natriuretic peptide 1913 ng/L (normal =125 ng/L). Portable ultrasonography showed small bilateral pleural effusions, but echocardiography was not available.

NS1 (nonstructural protein 1) dengue rapid test was positive and doctor decided to deliver the baby that day (illness day 4) in view of the likelihood of worsening thrombocytopenia and coagulopathy during the critical period of dengue. A baby girl weighing 2·22 kg was delivered by emergency caesarean section; she needed only supportive care until discharge from the neonatal unit 4 weeks later. The mother, who had been intubated during surgery, returned to ICU and needed a further 24 hour of BiPAP. Her platelet count reached a nadir of 25×10^9/L on illness day 6, but she had no significant bleeding or shock.

She had IgM seroconversion and real time PCR confirmed DENV-4. Throat swab was negative for influenza A and influenza B. Bronchoalveolar lavage and blood cultures were negative. Thyroid function was normal and autoimmune screen was negative. She was well enough to be discharged 8 days after admission, with a diagnosis of acute cardiac failure due to dengue myocarditis. At follow up 4 weeks later, she had a normal ECG and echocardiogram.

Dengue is one of the fastest spreading viral infections and 2·5 billion people now live in endemic areas. Cardiac involvement in dengue can range from myocardial impairment and bradyarrhythmias to fulminant myocarditis. Dengue myocarditis can present at any time during the illness, unlike other severe manifestations that present during the critical phase around defervescence. Cardiac effects have rarely been reported in pregnant women with dengue, which might be due to under reporting because of limited diagnostic methods in endemic areas, or misdiagnosis. Dengue is associated with poor maternal and fetal outcomes, but the contribution of cardiac morbidity has not been defined.

Reference: *The Lancet, 28 December, 2015; Vol. 385*

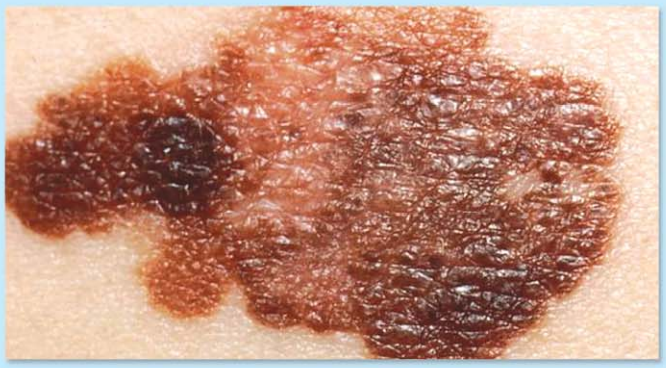
Info Quiz Answers (October-December 2015)

1. a **2. c** **3. c** **4. a** **5. d** **6. b** **7. e** **8. a** **9. d** **10. e**

HEALTH NEWS

Arm mole count predicts skin cancer risk

Research suggests that having more than 11 moles on one arm indicates a higher than average risk of skin cancer or melanoma.



Moles are small coloured spots on the skin made up of cells called melanocytes, which produce the colour (pigment) in the skin. They are long lasting and are not directly linked to sun exposure, but excess sun exposure will increase the risk of skin cancer and can make a mole turn malignant. Counting moles on the right arm was found to be a good indicator of total moles on the body. More than 100 indicates five times the normal risk. Researchers from King's College London studied a large group of female twins over a period of eight years, collecting information on skin type, freckles and moles on their bodies.

Reference: *www.bbc.com/health*

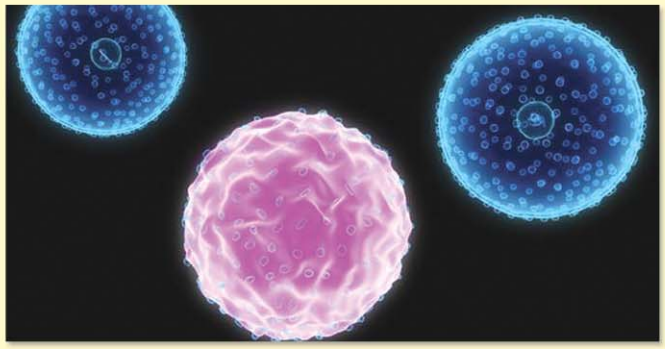
'Digital skin' activates brain cells

Engineers have built a flexible sensor that detects touch and, just like skin, produces electrical pulses that get faster when the pressure increases. The main advantage is that the plastic based sensor directly produces a pattern of pulses that makes sense to the nervous system. They are very thin and flexible, and are also stretchy. So it could mount a sensor on the skin and use it to detect vital signs like heartbeat and blood pressure. This is the first step towards using plastic materials for artificial skin on prosthetic limbs. The system is a more faithful replica of touch sensation than many other designs for artificial skin, making it a promising option for the development of responsive prosthetics.

Reference: *www.bbc.com/health*

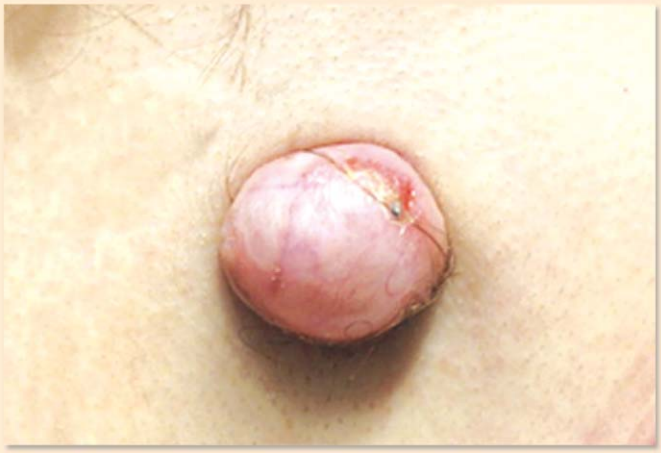
Leukemia cells can kill each other, study finds

Scientists at The Scripps Research Institute (TSRI) say the surprise finding, which involves changing leukemia cells into leukemia killing immune cells, could lead to a powerful new therapy for leukemia and possibly other cancers. Senior investigator Prof. Richard A. Lerner describes it as a "totally new approach to cancer." The Lerner laboratory has pioneered techniques to generate and screen very large libraries of antibodies (immune system molecules), in a search for therapeutic antibodies that bind to a desired target or activate a desired receptor on cells. Lerner and his team were testing 20 recently discovered receptor activating antibodies against acute myeloid leukemia cells from human patients, when one of these antibodies had an extraordinary impact on the acute myeloid leukemia cells. When the antibody was applied to healthy, immature marrow cells, it caused them to mature into blood platelet producing cells called megakaryocytes.



Reference: *www.medicalnewstoday.com*

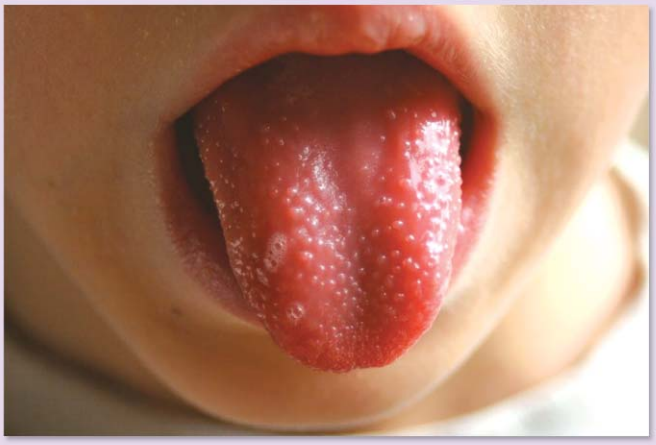
Cervical meningocele



A healthy 22 year old woman presented with a posterior cervical midline cutaneous lesion that had been present since birth and had been growing for years. The patient had undergone repeated aspirations, with no history of meningitis or myeloradiculopathy. Physical examination revealed a partially spherical lesion that was 2.5 cm in diameter, compressible, and non tender, with no active leakage. Neurologic examination was significant for Hoffmann's sign bilaterally and evidence of an upper motor neuron lesion affecting shoulders, arms, forearms, and hands. Magnetic resonance imaging revealed a fluid filled, dorsal cutaneous lesion and dorsal spinal cord tethering, with a connecting tract. Cervical meningoceles are uncommon neural tube defects. The recognition of spinal dysraphic conditions in adults can prevent infectious and neurodegenerative sequelae. The patient under went posterior cervical exposure of the lesion and spine, laminectomy, fibrous stalk resection, spinal cord detethering, and expansile duraplasty. She had no neurologic deterioration or cerebrospinal fluid leak in the hospital and was discharged home. At last follow up, she had no visible recurrence of the cutaneous lesion and no evidence of neurologic evolution of cervical myelopathy or radiculopathy.

Reference: *N. Eng. J. Med. 23 July, 2015; Vol. 373, N (4)*

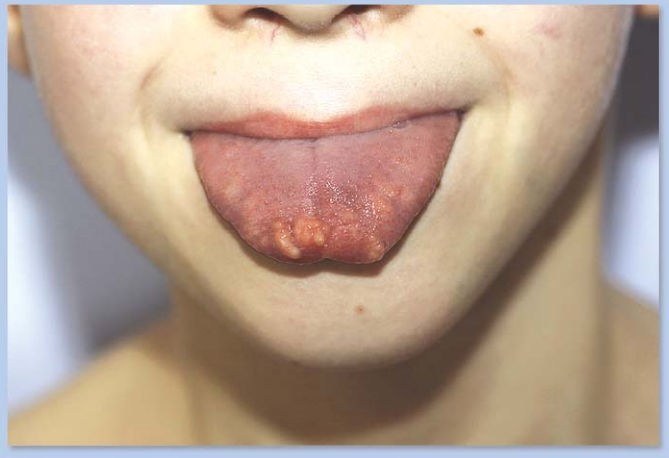
Strawberry tongue



An 8 year old girl was admitted to the hospital with seven day history of fever and rash. She had a diffuse macular rash, fissured lips, and strawberry tongue, and her temperature was 39°C. She had non exudative conjunctival injection in both eyes, a few enlarge cervical lymph nodes, erythema of the palms and sole, mild edema of the hands, and periungual desquamation. The cardiac examination was normal. A clinical diagnosis of Kawasaki's disease was made. Laboratory investigation revealed leukocytosis, with a white cell count of 13,000 per cubic millimeter (reference range, 3500 to 10,500) and an elevated erythrocyte sedimentation rate of 80 mm per hour (reference range, 0 to 29). The platelet count and results on electrocardiography and echocardiography were normal. She was treated with aspirin and immune globulin on admission and at a follow up visit 8 weeks later. Kawasaki's disease is a vasculitis of childhood. It occurs most frequently in children younger than 5 years of age and typically affects medium sized arteries. Accurate diagnosis and early therapeutic interventions can decrease the risk of coronary artery abnormalities. There is no diagnostic test that is specific for Kawasaki's disease; diagnosis is based on characteristic clinical findings and the exclusion of other possibilities in the differential diagnosis, including other infectious exanthems of childhood and reactions to drugs.

Reference: *N. Eng. J. Med. 30 July, 2015; Vol. 373, N (5)*

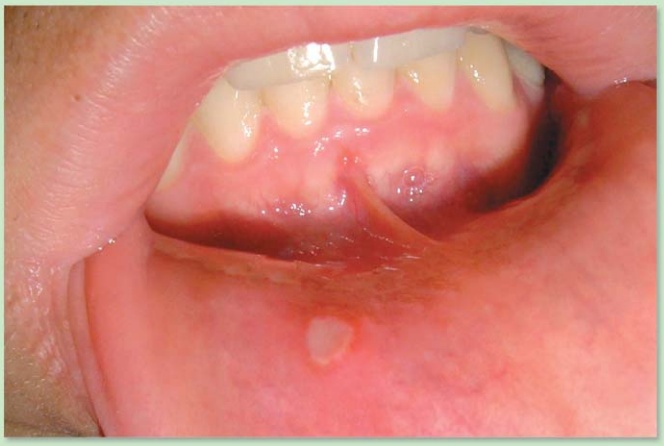
Multiple mucosal neuroma



A 9 year old boy presented with a 6 year history of asymptomatic papules on his tongue, along with chronic constipation and abdominal discomfort since infancy. On physical examination, he was noted to have a marfanoid habitus, coarse facies, tooth malposition, and multiple soft, yellow papules measuring 3 to 12 mm on the tip of his tongue. Findings on histologic analysis of samples obtained from the tongue lesions were consistent with mucosal neuroma and supported the diagnosis of multiple endocrine neoplasia type 2B (MEN2B). Thyroid ultrasonography revealed two hyperechoic nodules. Computed tomography of the abdomen showed large colonic distention. Laboratory evaluation revealed a calcitonin level of 277 pg per milliliter (normal value <10) and normal urinary and plasma levels of metanephrines. A de novo mutation in the gene encoding ret protooncogene (RET) was found. The patient underwent total thyroidectomy and nodal dissection that revealed bilateral medullary thyroid carcinoma with multiple metastatic lymph nodes. Rectal biopsy samples revealed intestinal ganglioneuromatosis. To date, the patient, whose disease is in remission, is under clinical surveillance with testing of serum calcitonin levels. Multiple lingual neuromas are suggestive of MEN2B and should prompt a careful evaluation for medullary thyroid carcinoma.

Reference: *N. Eng. J. Med. 20 August, 2015; Vol. 373, N (8)*

Oral manifestation of Crohn's disease



A 13 year old boy presented with a 9 month history of episodic unilateral swelling of the face and oral pain. He reported having loose, non bloody stools. Physical examination revealed asymmetric swelling of the face and lips with perpendicular fissuring, and intraoral examination revealed discrete gingival erythematous hyperplasia and epulis fissuratum like soft tissue tags in the mucobuccal fold. Granulomatous inflammation consistent with Crohn's disease was found on histopathological examination, and the patient was referred to a pediatric gastroenterologist. He was found to have tenderness to palpation in the right lower quadrant and periumbilical region, a rectal fissure, and painless rectal skin tags. Colonic biopsies showed chronic active colitis that was most prominent in the cecum and ascending colon, which confirmed a diagnosis of Crohn's disease. Therapy with mesalamine and prednisone was initiated and slowly tapered. Maintenance of remission was achieved with mercaptopurine. The oral lesions slowly resolved over a 1 year period. At a follow up visit 2 years after the initial presentation, the patient remained asymptomatic.

Reference: *N. Eng. J. Med. 24 September, 2015; Vol. 373, N (13)*

Bell's Palsy

Bell's palsy is a paralysis or weakness of the muscles on one side of the face, which is more common in 15 to 45 years of age. The facial nerve services the muscles of the face, the ear, salivary and tear glands, and provides some of the sensations of taste on the tongue. This nerve enters the skull via a small opening in the petrous temporal bone at the base of the skull. In Bell's palsy, the facial nerve swells and the resulting inflammation disrupts the relay of nervous system messages. The paralysis can be partial or total. It is thought that the inflammation and swelling of the facial nerve is caused by some type of viral infection or autoimmune system response. Bell's palsy is characterised by a droopy appearance around the eye and mouth on the affected side of the face. It is caused by swelling of the facial nerve at the point where it passes through a small opening in the skull. The pinched and swollen nerve becomes inflamed, which interferes with the nerve's proper functioning. Bell's palsy usually resolves by itself within a few months. Early treatment with corticosteroids may reduce severity.

History of discovering Bell's palsy

Bell's palsy is also called facial palsy. Bell's palsy is named for Dr. Charles Bell, a 19th century surgeon from Scotland who first described the condition.

Cause

The cause is still unclear, and its development is not well understood. However, some causes are given below:

- Viral infection - Herpes simplex virus
- HIV infection
- Vascular ischemia
- Autoimmune inflammatory disorders
- Heredity

There are some predisposing factors which also aid to create this disease. These are:

- Pregnancy
- Hypertension
- Diabetes
- Lymphoma

Symptom

- Sudden onset of paralysis or weakness on one side of the face, making it difficult to smile or close the eye on the affected side
- Facial stiffness or a feeling that face is being pulled to one side
- Pain behind or in front of the ear on the affected side
- Sounds that seem louder on the affected side
- Headache
- Facial droop and difficulty with facial expressions moves (inability to smile, frown, or whistle)
- Loss of taste on the front portion of the tongue
- Change in the amount of tears and salivary product

Diagnosis

Physical examination

The first step in diagnosis is to determine whether facial weakness is due to a problem in the central nervous system or one in the peripheral nervous system. Central weakness of the unilateral lower facial area, which is always due to a lesion above the level of the facial nucleus in the pons of the contralateral hemisphere, is explained by the fact that cells of the facial nucleus that innervate the lower face receive corticobulbar fibers primarily from the contralateral cerebral hemisphere. In contrast, cells of the facial nucleus that innervate the upper face receive corticobulbar fibers originating from both cerebral hemispheres. Thus, a unilateral lesion in the cortex or underlying corticobulbar fibers usually produces contralateral voluntary central type facial paralysis and a contralateral hemiplegia but does not affect salivary and lacrimal secretions or the sense of taste (Table 1).

Peripheral facial palsy, or a weakness or paralysis of all muscles of facial expression, is usually due to a lesion of the ipsilateral facial nerve but can also be produced by a lesion of the ipsilateral facial nucleus or facial nerve in the pons. Although it appears paradoxical that a "central" lesion in the pons produces peripheral facial weakness, the nomenclature is not likely to change. Facial weakness is best demonstrated by the patient's response to the

requests "Close your eyes" (for testing the upper facial area) and "Show me your teeth" (for testing the lower facial area). Denervation of the orbicularis oculi muscles will result in the inability of the patient to close the eyelids effectively, and denervation of the risorius muscle will result in limited retraction of the angle of the mouth.

Table 1. Clinical and anatomical features of facial nerve damage

Site of damage	Facial nerve signs	Associated features
Cortex, subcortical region	Contralateral central facial weakness, lacrimation, salivation, and taste intact	Contralateral hemiparesis and spasticity
Pons	Ipsilateral peripheral facial weakness, lacrimation, salivation, and taste intact palsy, ophthalmoparesis	Contralateral hemiparesis, sensory loss, ataxia, nystagmus and ipsilateral abducens
Cerebellopontine angle	Ipsilateral peripheral facial weakness, lacrimation, salivation, and taste usually intact	Tinnitus, facial numbness, ataxia and nystagmus
Facial nerve in internal auditory canal proximal to or involving geniculate ganglion	Ipsilateral peripheral facial weakness, lacrimation, salivation, and taste likely to be involved	Tinnitus, nystagmus and hearing loss
Facial nerve distal to internal auditory canal and geniculate ganglion	Ipsilateral peripheral facial weakness, lacrimation intact but salivation and taste impaired	Tinnitus, nystagmus and hearing loss
Facial nerve in stylomastoid foramen	Ipsilateral peripheral facial weakness, lacrimation, salivation, and taste intact	Head injury and parotid mass

Electromyography (EMG)

It confirm the presence of nerve damage and determine its severity. An EMG can measure the electrical activity of a muscle in response to stimulation and the nature and speed of the conduction of electrical impulses along a nerve.

Brain imaging

Magnetic resonance imaging (MRI) or computed tomography (CT) may be needed on occasion to eliminate possible sources of pressure on the facial nerve, such as an infection, tumor or skull fracture.

Management

Most patients with Bell's palsy (up to 85%) improve spontaneously within a few weeks. However, the after effects in the remaining 15-40 % can be so severe and distressing, that active treatment is essential. Most patients recover fully with or without treatment. Courses of treatment may include:

Anti-inflammatory medication: A short course of prescription corticosteroid medication, such asprednisone, may reduce inflammation and swelling in the narrow, bony channel through which the facial nerve travels.

Antiviral medication: Prescription antiviral medications, such as acyclovir (Zovirax) and famciclovir (Famvir), may limit or reduce damage to the nerve from some viral causes.

Physical therapy: Facial massage may help prevent permanent contractures of the paralyzed muscles before recovery takes place.

Beside these, eyes of the patient should be protected from damage to its outer layer (cornea) because of the absence of blinking on the side of the face with weakness. This can be done by artificial tears, an eye patch, transparent eye shield attached to the face with tape.

Complication

People who suffer long term from Bell's palsy, following complication may occur:

- Eye drying and corneal ulceration
- Facial weakness
- Speech problems
- Facial tightness (contracture)
- Loss or reduced sense of taste

References: *1. N. Eng. J. Med. 23 September, 2004; Vol. 351, N (11)*
2. www.betterhealth.vic.gov.au

Refresh your memory

Please select the correct answer by (✓) against a, b, c, d, e of each question in the Business Reply Post Card and sent it through our colleagues or mail within 17 February 2016; this will ensure eligibility for the Raffle Draw and the lucky winners will get attractive prizes!

1. In which indication Gelle's test is done?

a. Presbycusis
b. Serous otitis media
c. Otosclerosis
d. Meniere's disease
e. Multiple Myeloma

2. In which condition, subconjunctival haemorrhage is seen in children?

a. Whooping cough
b. Measles
c. Influenza
d. Chicken pox
e. Meningitis

3. Which drug will not be effective against Chlamydia, in case of treating a patient with active trachoma?

a. Azithromycin
b. Ivermectin
c. Rifampicin
d. Erythromycin
e. Cycloserin

4. What type of organism is Moraxella lacunata?

a. Gram negative diplococci
b. Gram positive diplococci
c. Gram positive diplobacilli
d. Gram negative rods
e. Gram negative diplobacilli

5. During normal involution, when uterus becomes a pelvic organ?

a. By the end of first week
b. By the end of second week
c. By the end of fourth week
d. By the end of sixth week
e. By the end of eighth week

6. Which of the following is not a feature of severe pre-eclampsia?

a. BP 160 /110 mmHg
b. Visual disturbances
c. Oliguria
d. Convulsions
e. Altered mental status

7. For ultrasound diagnosis of chronic polyhydramnios, which amniotic fluid index should be appropriate?

More than

a. More than 6 cm
b. More than 12 cm
c. More than 18 cm
d. More than 25 cm
e. More than 20 cm

8. Which of the following 5-HT agonists is anti anxiety drug?

a. Cisapride
b. Renzapride
c. Rizatriptan
d. Triptans
e. Buspirone

9. Which is not vapour phase disinfectants?

a. Ethylene oxide
b. Thiomersal
c. Formaldehyde gas
d. Betapropiolactone (BPL)
e. Propylene oxide

10. In which condition Warthin Finkedly cells are seen?

a. Warthin Tumor
b. Wolman disease
c. Whooping cough
d. Measles
e. Tetanus


ACI
ADVANCING
POSSIBILITIES