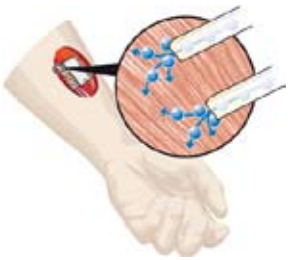
Trends & Technology

Integrated Chemical Chip Could Control Muscle Movement



A new integrated chemical circuit could lead to an array of new applications in life sciences, as it could be used *in vivo* to regulate the signal paths of cells in the human body. Developed by Klas Tybrandt, doctoral student researching organic electronics at Linköping University (Sweden), the circuits transmit ions and molecules. The chip can work with common signaling substances such as acetylcholine. Potential applications include sending signals to muscle synapses with a malfunctioning signaling system. Tybrandt and Berggren began to develop ion transistors three years ago that can control and transport ions and charged biomolecules. That research was published in 2010 in *Proceedings of the National Academy of Sciences* in an article titled "**Ion bipolar junction transistors**."
http://www.nature.com/ncomms/journal/v3/n5/full/ncomms1869.html

Needle-free jet injection

Needle-free drug delivery by jet injection is achieved by ejecting a liquid drug through a narrow orifice at high pressure, thereby creating a fine high-speed fluid jet that can readily penetrate skin and tissue. A controllable jet injection device, based on a custom high-stroke linear Lorentz-force motor that is feed-back controlled during the course of an injection.
The speed of the drug jet, injection depth (up to 16 mm) and precise volume (up to 250 μL) of drug delivered can be regulated. It may still take a few years before this or a related system is used widely, but there is hope that this type of technology will significantly reduce the dangerous number of occupational needle-stick injuries (estimated at over 385,000 in the US per year) as well as improve patient medication compliance.
http://www.sciencedirect.com/science/article/pii/S1350453311003249

Secure IV Catheter System

Tangent Medical out of Ann Arbor, MI recently unveiled its NovaCath Secure IV Catheter System, a device designed to improve peripheral IV catheter stabilization and reduce the potential for clinicians being exposed to patient blood. The closed system includes the catheter, high pressure extension tubing, multi-use flow control clamp and stabilization components. Designed to improve patient comfort and reduce overall IV complications including dislodgement, infiltration, phlebitis, and occlusion.

http://medgadget.com/2012/05/tangent-shows-off-novacath-secure-iv-catheter.html

Smiths Medical's ViaValve Safety I.V.

ViaValve Safety I.V. Catheter promises to help prevent blood exposure and needlestick injuries, which, each year, affect approximately 800,000 hospital-based healthcare workers in the U.S. by means of an integrated valve in the catheter hub. The valve provides blood control by impeding the backflow of blood from the patient's vein upon initial venipuncture. ViaValve™ Safety I.V.

Ultrasonic fog machine kills superbugs

Altapure LLC has launched a portable model of its ultrasonic sterilization device that kills pathogens in large areas like hospital operating and patient rooms. The machine's technology, creates pressure waves at the surface of common liquid cleaning agents like peracetic acid or hydrogen peroxide and disperses a thick fog made up of sub-micron-sized droplets that spreads quickly throughout a room, clings to surfaces and eradicates harmful pathogens in seven to 10 minutes, a 100 percent kill rate in testing with medical-grade spores of geobacillus stearothermophilus and bacillus atrophaeus. The new model, the HJ-30i, delivers major breakthroughs in size, weight, performance flexibility and scalability.
Altapure is located in South Bend, Indiana at Innovation Park.

A new rapid Bloodless glucometer

Grove Instruments' Optical Bridge technology uses near-infrared spectroscopy to measure a person's real-time blood sugar in less than 20 seconds. The company's first product is an accessory-free, battery-operated personal glucose meter used on the fingertip or earlobe. Grove is one of several companies working on a noninvasive diabetes test using spectroscopy includingDIRAmed, C8 MediSensors and InLight Solutions. Challenges in developing devices using this technique have included water interference and low signal-to-noise ratio, but Grove thinks it has developed solutions to these problems.

CLINIQUIZ

Arterial blood gas analysis

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Arterial blood gas estimation is now an established diagnostic modality for evaluation of altered homeostasis in critically ill patients. The current questionnaire focuses on case scenarios commonly seen in anaesthetic and critical care practice, where accurate interpretation plays a crucial role in their management. (Please choose one best option).

Q 1: A 40 yr old male, presented to the emergency department with a history of fever, cough, expectoration and shortness of breath. On examination, the case had BP: 190/114 mmHg and fine inspiratory crepitations at both lung bases. The blood gas values (on room air) were: pH 7.76, $PaCO_2$ 20 mm Hg, HCO_3^- 28 mmol/L. These findings are suggestive of;
a. Mixed metabolic and respiratory acidosis
b. Mixed metabolic and respiratory alkalosis
c. Mixed metabolic acidosis and respiratory alkalosis
d. Mixed metabolic alkalosis and respiratory acidosis

Q 2: Which of the following statement is incorrect regarding blood collection procedure for ABG analysis;
a. Arterial puncture should not be performed distal to arteriovenous fistula
b. Heparinization of sample may alters the value of pH
c. Heparinization of sample may alters the value of PaCO2/ PaO2
d. Delay in running the sample decreases PaO2

Q 3: In order obtain accurate blood gas results, all of the following parameters need to be entered in ABG machine except;
a. Patient temperature
b. Haemoglobin
c. Humidity
d. Barometric pressure

Q 4: Which of the following is the major buffer system in the extracellular fluid (ECF);
a. Ammonium
b. Proteins
c. Bicarbonate
d. Phosphate

Q 5: Which of the following statement is incorrect regarding the maximal limit of compensation in an acid-base disorder;
a. Metabolic acidosis ($PaCO_2$ lower limit: 5 mm Hg)
b. Metabolic alkalosis ($PaCO_2$ upper limit: 60 mm Hg)
c. Chronic Respiratory alkalosis (bicarbonate lower limit:12-15 meq/L)
d. Acute Respiratory alkalosis (bicarbonate lower limit:18 meq/L)

Q 6: A 30 yr old male, known diabetic (IDDM), presented to the emergency department with acute shortness of breath. On examination, he had tachycardia and was obtunded. The ABG results (on 80% oxygen by non Rebreathing mask with reservoir) were: pH 7.08, $PaCO_2$ 31 mm Hg, HCO_3 9.0 meq/L, PaO_2 54 mm Hg, Anion Gap 21 (Na^+ 137.6 meq/L, K^+ 2.4 meq/L, Cl^- 110 meq/L). These findings are indicative of;
a. Mixed Metabolic and respiratory acidosis with moderate hypoxemia
b. Mixed Metabolic and respiratory alkalosis with mild hypoxemia
c. Mixed Metabolic acidosis and respiratory alkalosis with severe hypoxemia
d. Mixed Metabolic alkalosis and respiratory acidosis with moderate hypoxemia

Q 7: The type of metabolic acid-base disorder in this case is best quantified by;
a. High anion gap metabolic acidosis
b. Non anion gap metabolic acidosis
c. Both High and Non anion gap metabolic acidosis
d. None of the above

Q 8: Which of the following combination is not possible in a triple acid –base disorder;
a. Metabolic acidosis, metabolic alkalosis and respiratory acidosis
b. Metabolic acidosis, respiratory alkalosis and respiratory acidosis
c. Metabolic acidosis (both high and normal anion

gap) and respiratory alkalosis
d. Metabolic acidosis (both high and normal anion gap) and respiratory acidosis

Q 9: The patient is resuscitated and after 2 hr of treatment the ABG results were: pH 7.04, $PaCO_2$ 60 mm Hg, HCO_3 16 mmol/L, Anion gap 12 (Na^+ 135 meq/L, K^+ 2.0 meq/L, Cl^- 109 meq/L). These findings are suggestive of treatment with one of the following agents;
a. Furosemide
b. Sodium Bicarbonate
c. Rx with ventilatory support
d. Normal Saline

Q 10: Which of the following is the most probable cause of post-resuscitation worsening of acidosis and hypokalemia in this case;
a. Furosemide
b. Sodium Bicarbonate
c. Rx with ventilatory support
d. Normal Saline

ANSWERS

Ans 1: (b) Mixed metabolic and respiratory alkalosis. Using the arithmetic equation for metabolic alkalosis (predicted PaCO2 = 0.7*bicarb value + 21 ±2), the PaCO2 is expected to be 40.6 mm Hg. As observed PaCO2 is lower in this case, co-existing primary respiratory alkalosis is also present.

Ans 2: (b) Excessive heparinization leads to blood dilution resulting in decline of PaCO2 and rise in PaO2 values. However, pH variation does not occur (even after 50% dilution by heparin) because of vast buffering effect of haemoglobin and plasma proteins present in the blood. Sample delay leads to consumption of oxygen present in blood with consequent decline in PaO_2. It is contraindicated to perform arterial puncture distal to AV fistula, as it may precipitate unopposed bleeding from the dilated hypertrophied vessels; indeed it preferable to draw arterial samples from other extremity.

Ans 3: (c) For obtaining the blood gas results, patient temperature, haemoglobin, FiO2, and barometric pressure (P_b) needs to be provided in the ABG machine. ABG machine then calculates the PaO_2 values using the arithmetic equation: PaO_2 = PiO2–1.2(PaCO2) and PiO2 = FiO2 (P_b–47)]. Routinely machine analyzes the blood sample at 37°C. If the fed temperature is 37°C in a sample of hypothermic patient, measured values of PaO_2 and PaCO2 will be more than actual data and vice-versa.

Ans 4: (c) Bicarbonate system accounts for 80% of buffering in the ECF. On chemical grounds, it (pKa 6.1) should not be a good buffer in comparison to phosphate (pKa 6.8). However, higher concentration of bicarbonate in ECF along with its ability to regulate $PaCO_2$ via lungs greatly enhances the buffering capacity of this system. Phosphate and proteins are the important buffers in intracellular fluid and urine. Ammonium is predominantly produced in proximal tubules of kidney making it an important buffer in urine only.

Ans 5: (a) The lowest level to which $PaCO_2$ (respiratory compensation) can fall in metabolic acidosis is typically 8-10 mm Hg. As calculated by arithmetic equation (predicted $PaCO_2$ = 1.5 x Bicarbonate value + 8 ±2), bicarbonate values needs to be negative for getting a $PaCO_2$ < 8 mm Hg, which is not possible clinically.

Ans 6: (a). This disorder is primarily of metabolic origin (acidosis) as signified by the low values of bicarbonate. Using the arithmetic equation for metabolic acidosis (predicted PaCO2 = 1.5*bicarb value + 8 ±2), the PaCO2 is expected to be 21.5 mm Hg. As observed $PaCO_2$ is higher in this case, co-existing primary respiratory acidosis is also present. The PaO_2 value of 54 mm Hg is indicative of moderate hypoxemia.

Ans 7: (c) In this case, anion gap value of 21 is indicative of high anion gap metabolic acidosis. However, using the arithmetic equation [corrected bicarbonate = observed bicarbonate + (anion gap-12)], corrected bicarbonate comes out to be 18. As this value is less than 24, coexisting non anion gap metabolic acidosis is also present. Thus, triple acid base disorder exists in this case.

Ans 8: (b) Triple acid-base disorder is present when a respiratory acid-base disorder occurs in conjunction with a co-existing mixed metabolic disorder. Mixed respiratory disorder (both respiratory acidosis and alkalosis) is not possible as CO2 can never be both over- and under-excreted by the lungs at the same point of time.

Ans 9: (b) Such a steep rise in plasma bicarbonate level is most likely due to treatment with sodium bicarbonate. Furosemide exhibits weak carbonic anhydrase activity and increases bicarbonate excretion while ventilatory support results primarily in steep decline in $PaCO_2$ (not evident) followed by slow decline in bicarbonate levels. Infusion of normal saline would have resulted in hyperchloremia not evident in this case.

Ans 10: (b) Initial respiratory component in this case was most likely due to the respiratory muscle weakness as a result of hypokalemia. Bicarbonate administration as an initial therapy resulted in intracellular shift of potassium ions leading to worsening of hypokalemia and consequent respiratory muscle weakness. Thus, worsened respiratory acidosis (elevation in PaCO2) neutralized any buffering effect of bicarbonate with an aggravated acidemia. Appropriate treatment strategy in this case should be bicarbonate administration along with slow replacement of body potassium and invasive ventilatory support.

REFERENCES

1. M D Dake, J Peters, and R Teague. The effect of heparin dilution on arterial blood gas analysis. West j med. 1984; 140(5):792-3.
2. Shrake K, Blonshine S, Brown R, Ruppel G, Wanger J, Kochansky M. AARC Clinical Practice Guideline: Sampling for Arterial Blood Gas Analysis. Respiratory Care 1992(8); 37 891–7.
3. Zimmerman JL, Dellinger RP. Blood gas monitoring. Crit Care Clin. 1996; 12(4):865-74.
4. Blonshine S. Arterial Blood Gas Analysis. AARC Times 1999; 55-7.
5. Williams AJ. Assessing and interpreting arterial blood gases and acid-base balance. BMJ 1998; 317:1213.

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