The CNYPCC Toxicology Letter

Introduction
The Central New York Poison Control Center (CNYPCC) is pleased to provide you with the first issue of “The CNYP Toxicology Letter”. Published quarterly, this newsletter will be distributed to all the Emergency Departments and Critical Care Units within our 14 county service area. Designed for healthcare professionals, the letter will discuss issues specifically directed to the evaluation and management of the poisoned patient. As further issues are generated, we hope to encourage suggestions from our audience on design and content. To that end, please feel free to contact the CNYPCC at the address and phone number listed at the end of this newsletter.

CASE HISTORY

ANESTHESIA EMERGENCY

A 29 year old female complaining of lower back pain is to receive a trigger point injection of 10 mL of 0.75% bupivacaine. A needle was inserted, and after a negative aspiration for blood the drug was administered. After 1-2 minutes, she complained of nausea and 2 minutes later she had a witnessed cardiac arrest. Standard advanced cardiac life support with cardiopulmonary resuscitation was initiated, and after a prolonged resuscitation, the patient was stabilized.

What is bupivacaine?
Bupivacaine is a local anesthetic of the amide class. Examples of other local anesthetics of the amide class include mepivacaine and lidocaine. Bupivacaine has a long duration of action, with the onset of nerve block occurring in 4-7 minutes and lasting for 4-7 hours. It is highly protein bound and is eliminated principally through hepatic metabolism. Bupivacaine is used for peripheral, sympathetic and epidural block anesthesia.

What is the toxicity from bupivacaine and how does it compare to mepivacaine and lidocaine toxicity?
Toxicity from all local anesthetics includes both cardiac and central nervous system effects. These effects are due to a high local concentration of the drug, and generally occur only after a high dose of the drug has been inadvertently given or a therapeutic dose of the drug has been given too quickly. Cardiac effects are due to the drug’s class IB antidysrhythmic effect which slows phase 4 automaticity and may result in sinus arrest, AV block, hypotension, ventricular dysrhythmias and cardiac arrest. Central nervous system effects include drowsiness, weakness, euphoria, dysphoria, paresis, muscle fasciculations and seizures. Lidocaine toxicity typically results in seizures activity before the onset of its myocardial depressant effects, whereas bupivacaine and mepivacaine may produce cardiac effects simultaneously with CNS effects. Also, as is the case with this patient, cardiac effects of bupivacaine may occur without any CNS effects at all.

If a patient is allergic to one local anesthetic are they allergic to all of them?
There are currently two types of local anesthetics available; the ester containing and the amide containing local anesthetics. The allergy associated with amide anesthetics (i.e. lidocaine, bupivacaine, mepivacaine) is generally due to the drug’s preservative. True allergy to the amide drugs is possible, however it is rare and limited to the specific agent responsible.

Continued on back page
Continued from front page

Therefore, a patient with an allergy to one amide can safely be given either another amide local anesthetic or an ester anesthetic if it does not contain the same preservative.

The ester anesthetics are different. Some of the agents included in this group are cocaine, procaine, benzocaine and tetracaine. These agents are metabolized to a common product, para aminobenzoic acid (PABA). Allergy to any of these agents is due to an allergy to this metabolite. Thus, after an allergic reaction to one agent in the ester class occurs, the use of another agent in this class is likely to result in cross-allergenicity. In these cases, it would be prudent to switch to an agent of the amide class for future local anesthetic use.

An easy way to remember these groups is that the common amide group local anesthetics contain two j's in their name where the common ester group local anesthetics contain only one j in their name.

How can we protect patients from receiving inadvertent intravascular administration of anesthetics?

Prior to the injection of a local anesthetic in a peripheral site, it is generally recommended to check for possible entry in a vascular structure by ensuring that there is no blood return on aspiration. This should be done in all cases, however, as in this case it may not be adequate for several reasons. First, aspiration in a small vein may temporarily collapse the vein, resulting in a negative aspiration. Also, a vein may become lacerated as a needle passes through it. This may result in a negative aspiration because the needle is actually in the vein, but the needle opening is outside the vein. The drug enters the vein through passage that is left after the needle is withdrawn.

How is local anesthetic toxicity treated?

This patient probably received an intravenous bolus of bupivacaine instead of a peripheral nerve injection. The resultant increased vascular concentration produced myocardial toxicity. Treatment available for these patients is limited. Seizures should be treated with supportive care and benzodiazepines. Cardiovascular toxicity should be treated with advanced cardiopulmonary resuscitation. The use of hyperventilation and/or sodium bicarbonate has been proposed for the treatment of sodium channel blockade. This may have some role and should be tried for cardiovascular effects induced by these agents.

The length of toxicity is dependent on the time the drug is in contact with the affected organs. Lidocaine redistributes to the periphery quickly and toxicity is usually limited to minutes. However, the cardiodepressant effects from other local anesthetics can be prolonged. Bupivacaine and mepivacaine induced cardiopulmonary arrest is usually long lasting and prolonged resuscitation measures and bypass are often required and are still frequently unsuccessful. When patients survive, they occasionally are left with neurological deficits. This patient was unfortunately left with severe neurological defects requiring ventilatory support.

REFERENCES


TOX TRIVIA:

1. What is the #1 toxin related cause of death in the United States?
2. What is the toxic effect of shoe desiccants (silica gel)?
3. What is the toxin that is contained in jimson weed?
4. Why is freezing point depression the preferred method for determining osmolality in toxic alcohol poisoned patients?

CLINICAL TOXICOLOGY PEARLS:

1. Any salicylate exposed persons urine will turn violet purple when a few drops of 10% ferric chloride solution is added to it.
2. Fatal hemoglobin reads as carboxyhemoglobin on a co-oximeter.
3. The blood of a patient with methemoglobinemia will appear brown.

CNY POISON CONTROL CENTER • 750 EAST ADAMS STREET, SYRACUSE, NY 13210 • 315-476-4766
**AM I BLUE?**

A ten month old previously healthy infant presents with cyanosis. His parents state that over a four hour period, while at play, they noted him to become “bluer and bluer.” He has been well during the past few days, without cough, fever, change of behavior, or known contacts. His past medical history is essentially negative. He takes no medications. 

On presentation the infant is afibrile with a respiratory rate of 30. His heart rate is 120 and regular. There is no work of breathing noted. His capillary refill is two seconds. He has peripheral and central cyanosis. His blood pressure is 90/50. His lungs are clear and no murmurs are noted on exam. His abdomen is soft, non-tender, without mass. Full peripheral pulses are palpable. He is neurologically intact.

**What immediate interventions are indicated at this point?**

Since the infant is cyanotic, 100 percent oxygen by mask should be administered. In addition, the patient should be placed on a cardiac monitor to determine whether any arrhythmia is present. Intravenous access should be obtained as well. Peripheral oxygen saturation may be obtained by the use of an oximeter at the bedside.

**What is the differential diagnosis of cyanosis in infancy?**

When evaluating an infant with cyanosis out of the newborn period (0-28 days), the clinician is advised to consider the physiologic function of three organ systems, namely the cardiac, pulmonary, and hematologic systems. With regard to the likelihood of acute cyanotic pulmonary events, consideration should be given to the potential for foreign body aspiration, acute bronchospasm, or the presence of a spontaneous pneumothorax. In this case, however, the absence of significant respiratory distress diminishes the likelihood of these pulmonary entities.

Infants born with cyanotic cardiac lesions will most often present within the first ten days of life with cyanosis and/or respiratory distress. The five most common cyanotic cardiac lesions include Tetralogy of Fallot, Transposition of the Great Vessels, Truncus Arteriosus, Trisomy 22, and Total Anomalous Pulmonary Venous Return. Since this child's past medical history was unremarkable, it is unlikely that an unknown cyanotic cardiac lesion would present at 10 months of age.

The ability of hemoglobin to carry oxygen may be impaired in certain situations. Exposure to carbon monoxide in sufficient quantities will displace oxygen from the hemoglobin molecule. An additional impairment to the oxygen carrying capacity of hemoglobin would be a situation where the iron molecule within hemoglobin is changed from the ferrous to the ferric state, namely methemoglobinemia.

**How can the laboratory assist in making a diagnosis in this case?**

All patients presenting with cyanosis should undergo radiographic evaluation of the chest. In this scenario the chest film demonstrated a normal cardiac silhouette, with clear lung fields and the absence of the pneumothorax. An ECG should also be obtained to rule out structural anomalies of the heart. In our patient, the ECG was unremarkable. An arterial blood gas demonstrated a pH of 7.43, a PCO2 of 36, and a PO2 of 385. The oxygen saturation measured at the bedside was 90 percent. Precise measurement by a co-oximeter within the laboratory concomitant with the ABG revealed an oxygen saturation of only 70 percent. In addition, some drops of blood were spilled during the arterial puncture, which were brown in color, signifying the presence of methemoglobin.

**What is the diagnosis in this case?**

The scenario of cyanosis unresponsive to supplemental oxygen in the absence of cardiopulmonary compromise should alert the clinician to the presence of an abnormal hemoglobin oxygen transport system, i.e. methemoglobinemia. In addition, the conversion of red blood to a brown color when exposed to room air also points to this diagnosis as well.

**What is methemoglobinemia?**

Methemoglobinemia represents the clinical presentation of cyanosis and other findings consistent with asphyxia relative to the conversion of iron from the ferrous to ferric state. When converted, the iron is incapable of binding oxygen and therefore will not deliver oxygen to the periphery.

Methemoglobinemia may be caused by three essential mechanisms:

a. Hereditary presence of an abnormal hemoglobin (hemoglobin M).

b. Hereditary deficiency of naturally occurring methemoglobin reductase.

c. Exposure to oxidant drugs or chemicals.

Of the aforementioned entities, the last category represents the etiology of most methemoglobinemia cases.
TOX TRIVIA:

1. What was the name of the NSAID that was withdrawn from the pharmaceutical market due to a high incidence of anaphylactic reactions?

2. What is the odor that is associated with cyanide poisoning and what is the compound that produces the odor?

3. How much commercial ground nutmeg would be required to be ingested to cause psychoactive effects?

4. Etoposide is a synthetic analog of?

CLINICAL TOXICOLOGY PEARLS:

1. 6 hours non-toxic??? What clinical finding should be absent for this statement to be true for the following toxins? a. tricyclic antidepressants b. iron

2. If a patient is cyanide poisoned, the arterial blood gas will not improve after supportive care.

3. Sulfhemoglobin is read as methemoglobin on a co-oximeter. Add cyanide to sulfhemoglobin and the interference will be diminished.

REFERENCES


CNY POISON CONTROL CENTER • 750 EAST ADAMS STREET, SYRACUSE, NY 13210 • 315-476-4766
The CNYPCC Toxicology Letter

CNYPCC STAFF:
Richard Cantor, M.D., FAAP, FACEP, AACT, Medical Director
Christine Stork, Pharm.D., AACT, Director
Michele Caliva, R.N., Supervising Poison Information Specialist
Poison Information Specialists:
Carol Sopchak, R.N.
Debra Abert, R.N.
Laurie Piwniski, R.N.
Teesh Guenther, R.N.
Linda Juton, R.N.
Trudy Dody, M.S., R.N.
Mary Hollenbeck, R.N.
Kathleen Groff, R.N.
Nancy O’Neill, R.N.
Lynn Wilder, R.N.
Patricia Koniz, R.N.

EDUCATIONAL STAFF:
Gail Banach, M.S.
Kristi Newton, M.S.

CLERICAL STAFF:
Ja Juehlen

SCHEDULED EVENTS:
S.U.N.Y., H.S.C. Department of Emergency Medicine
Emergency Medicine Grand Rounds
Marley Education Center: Sulzle Auditorium
Third Friday of the Month, 11:00 AM
July 19
Pediatric Surgical Emergencies
August 16
Genitourinary Problems in the Emergency Department
September 20
CPR: Where We Were, Where We Are, and Where We Are Going

Toxicology Case Conference
CNYPCC, 550 E Genesee Street
Poison Center Conference Room
Every Thursday 11:00 AM - 12:00 PM

NEWS BULLETIN:
On April 10, 1996 the FDA issued a warning about the product “Herbal Ecstasy”. This product has been associated with over 100 reports of adverse effects, including 15 deaths. The active ingredient is ephedrine and may also be listed as Ma Huang, ephedra sinica or ephedra extract.

CASE HISTORY

VACATION EMERGENCY

A 29 year old female is brought to the Emergency Department with a 3 day history of increasing lethargy, weakness, difficulty swallowing and left hand paresthesias. Three days prior to admission, while vacationing in Florida, the patient swam in a “red tide” and had also eaten a large amount of Sheepshead and Yellowtail fish caught from the ocean. Approximately 12 hours after eating, she experienced shortness of breath, mild chest pain, and tingling left hand digits. These symptoms increased in severity until the day of admission where she was unable to lift her 15 month old daughter.

The patient’s past medical history is non-contributory. Physical examination reveals a well developed, well nourished woman in no apparent distress. Significant physical examination findings include a blood pressure 120/60 mm Hg, pulse 88 bpm, temperature 37.1 C and respiratory rate 20 bpm. There is mild generalized abdominal tenderness without rebound or guarding. On neurologic assessment, she is alert and oriented to person, place, and time. Reflexes are 2+ in the upper and 4+ in the lower extremities. Motor strength is 5/5 throughout. There is a decrease in left hand light touch perception in the distribution of C6 and C7. During the first day of hospitalization, the patient develops perioral tingling and tingling of the tongue. Gait was normal and no nystagmus was present. Laboratory tests include a Na 144 meq/L, Cl 90 meq/L, K 3.9 meq/L, HCO3 25 meq/L, Ca of 10 meq/L, P04 3.7 meq/L. Renal and hepatic function studies are normal. Also, uro acid, albumin, CBC, ESR and UA are all within normal limits. Chest radiograph, electrocardiogram and MRI are read as normal.

What is the patient’s differential diagnosis?
There are several causes of acute neurologic dysfunction. This patient gives a history of exposure to the “Red Tide” and to seafood that the patient obtained while fishing that same day. We will limit our discussion to toxins associated with her exposure.

What is the “Red Tide”?
During the non “R” containing months of the year (May through August) plankton proliferate through photosynthesis in warm areas of the world. This phenomena occurs on both the East and West coasts of the United States and generally occurs between 30 degrees North and South latitude of the equator. These plankton belong to the phylum Protozoa and are single celled pigmented organisms. Shellfish feed off these dinoflagellates and bioconcentrate their toxin. Subsequent ingestion of these shellfish may cause neurologic, paralytic or amnesic symptoms. If large numbers of dinoflagellates are present, walking down a beach that is infested with the red-tide may produce bronchospasm due to aerosolized toxin. The most frequently implicated dinoflagellates include Phycodiscus brevis [neurotoxic], Protogonyaulax axinella and Protogonyaulax tamarensis [paralytic], and Nitzschia pungens [amnesic]. The neurotoxic dinoflagellate produces the toxin brevetoxin, while the paralytic dinoflagellate produces saxitoxin. The amnesic shellfish poison is thought to be domoic acid. All the toxins associated with the red-tide are heat insensitive. The onset of symptoms associated with the red tide is usually within 30 minutes to several hours after exposure. Neurotoxic shellfish poisoning from brevetoxin may appear 15 minutes to 18 hours (usually 3 hours) after exposure as “hot-cold inversion of sense”, myalgias, nausea, vomiting, diarrhea, bradycardia, decreased reflexes and dilated pupils. Brevetoxin also increases sodium flux in bronceal mucosa and may result in bronchoconstriction. Symptoms usually resolve within a day after exposure with a range of 1 to 72 hours.

Paralytic symptoms from saxitoxin usually begin approximately 30 minutes after exposure and may include paraesthesia or extreme paraesthesias, headache and ataxia, which may progress to paralysis and cranial nerve dysfunction. Death is usually due to respiratory failure and occurs within 12 hours of exposure.

Amnesic shellfish poisoning may have a delay in onset of about 5 hours (15 minutes to 38 hours) and is heralded by gastrointestinal symptoms such as nausea, vomiting, and diarrhea followed by memory loss. Severe, more severe neurologic symptoms of coma, seizures, and hemiparesis may occur. The suspected toxin, domoic acid, may interact with NMDA receptors as it is a structural analog of glutamic and kainic acid. These excitatory amino acids are currently being researched for their possible role in causing neuronal cell death.

Treatment for red-tide poisoning is supportive, with special attention to airway and respiratory support. Gastrointestinal decontamination should be considered in symptomatic individuals. It is unlikely that our patient was poisoned through contact with the red-tide. Her exposure could have been to aerosolized toxin and should have resulted in respiratory symptoms which did not occur. Also, the timing of her symptoms was relatively late for brevetoxin, starting at 12 hours and lasting for 3 weeks. Upon questioning, we also learned that several other family members also went swimming in the red-tide without clinical effect so we considered this an unlikely etiologic agent for this patient’s symptoms.

Continued on back page
What is the differential of fishborne poisoning?

There are many toxins that are associated with the ingestion of fish products. The large differential can be narrowed by determining the type of fish ingested, the area in the world where the person obtained the fish and the signs and symptoms of toxicity that the patient is experiencing. Our patient was in Florida, caught her own fish and ate it. This excludes many exotic fish-borne illnesses that would be a consideration in other areas of the world. It does however, leave several options to consider.

Scombroid poisoning is a common type of fish poisoning that occurs with the improper refrigeration of fish. Fish most often associated with the production of toxin leading to scombroid poisoning include albacore, bluefin and yellowfin tuna, dolphinfish, sardine, anchovy, herring and bluefish. During periods of non-refrigeration, the musculature of these fish undergoes decomposition. Bacteria (classically *Morganella morganii*) converts the amino acid l-histidine to histamine and saurine (histamine hydrochloride). Affected fish may have a metallic or peppery taste. Symptoms usually develop within 15-90 minutes after exposure and include flushing, pruritus, urticaria, angioneurotic edema, bronchoospasm, nausea, vomiting, headache, tachycardia and hypotension. Treatment consists of supportive care and antihistamines. Outcome is generally favorable with symptoms resolving within 6 to 12 hours.

Tetrodotoxin is a fish poison within the specific fish order Tetraodontiformes. Examples of fish containing tetrodotoxin include the pufferfish (toadfish, blowfish, globefish, swellfish, balloonfish) and porcupine fish. Tetrodotoxin is thought to be identical to the toxin tetrachotoxin which is found in North American and Chinese newts, international salamanders, the skin of the Central American frogs (genus Ateles), some shellfish, the Ribbon worm, Flatworm, Horseshoe crab and the Blue crab. The toxin is an amino-pyridyloquinazoline and is found throughout the fish with highest concentrations in the liver, gonads, intestine and skin. The mechanism of toxicity is the blocking of sodium channels at the level of the axon. Symptoms after exposure to tetrodotoxin can occur in as little as 10 minutes, but can be delayed for up to 4 hours. Initially, the patient may present with perioral paraesthesia, lightheadedness, generalized paraesthesia, nausea, vomiting, weakness. An ascending paralysis may develop with death occurring in 6-24 hours. Other symptoms that may occur include seizures, hyperactivity, diarrhea, and ataxia. Mentation may be maintained despite flaccid paralysis. Survival past 24 hours is considered a good prognostic sign.

Ciguatera toxin is usually found in fish that live between the 35th degree North and South latitude. These fish are usually bottom dwelling Reef fish, and poisoning usually occurs in the spring and summer months. Large fish (greater than 6 pounds) and older fish bioaccumulate the toxin as they ingest smaller herbivorous fish that extract the dinoflagellate Gambierdiscus toxicus from blue-green algae. Many species are implicated in causing ciguatera poisoning. These include mullets, groupers, snappers, parrot fish, amberjack and barracuda. Symptoms of ciguatera occur within 1-3 hours after ingestion and include abdominal pain, nausea, vomiting, diarrhea, chills, parasthesias (perioral, classic "hot/cold" dissociation), a metallic taste, and pruritus. Rarely, the patient's course may progress to respiratory failure and death. The gastrointestinal symptoms generally resolve in 24-48 hours, but the neurologic symptoms may remain for several days to weeks, often correlating with the amount of toxin ingested.

HISTORICAL "MATCHING" TIDBITS:

1. Vitus Guerullias (Tennis Player) a. amyl nitrate
2. Paul Lynda (hollywood squares) b. CN
3. John Belushi c. speedball (heron/cocaine)
4. Jonestown Massacre d. CO

REFERENCES


Beware the "Fascinoma"

Case One: Factitious Hypoglycemia
A 2 year old male arrived in an unsupervised state. He was said to be unarousable after his regular afternoon nap. The staff were not able to arouse him. The patient was unresponsive. His physical examination revealed a comatosed with a table of purposeful responses. Vital signs were normal, with the exception of mild tachycardia. Pupils were midposition, equal and reactive. Fundus were normal. There was generalized increase in muscle tone with intermittent decerebrate posturing. Deep tendon reflexes were preserved. Laboratory analysis: blood glucose was 2.2. Electrolytes otherwise normal. Tests of urine, serum, and gastric aspirates were negative for salicylates, APAP, alcohol, and oral hypoglycemics. A skeletal survey showed a healed fracture of the left radius and ulna.

An IV was begun, a solution of D5W was given, and a continuous infusion of D5W begun. Interventions included augmented glucose support to D10W and steroids, with eventual normalization of blood sugar. By 20 hours, his anorexia improved with a fall in serum glucose levels. Full neurologic recovery was noted. Serum insulin and C-peptide levels drawn at 2, 4, and 12 hours, and at 2 and 4 months post discharge demonstrated a hyperinsulinemic state which had resolved after discharge from the hospital.

Additional history demonstrated the presence of the mother's boyfriend who was also an insulin-dependent diabetic. Full disclosure was obtained from the boyfriend after social services investigation.

Case Two: Chronic Diarrhea and Failure to Thrive
A 23 month old female was admitted for the third time with a chief complaint of vomiting and diarrhea. The child lived at home with her mother who was separated from the father. History was positive for 10 to 15 watery stools every day since infancy. The patient was pale and irritable, with growth parameters clearly demonstrating a decline from the 50th to 7th percentile for height. During each of her hospitalizations, the child had watery, pink tinged stools that averaged 250 grams per day.

Extensive laboratory analysis was normal for CBC, ESR, urinalysis, immune status, stool cultures, upper GI imaging, barium enema, urine catecholamines, and 72 hour fecal fat content. A small intestinal biopsy was normal as well.

Alkalization of multiple stools with sodium hydroxide showed a pink color change, suggesting the presence of phenolphthalein. The patient, when confronted with the cause of the patient's diarrhea, angrily denied giving laxatives. The child was placed in foster care, with close follow-up demonstrating normal catch-up growth and normal stool patterns.

Case Three: Recurrent Vomiting, Muscle Weakness, Rhythm Disturbances
A 16 month old male was evaluated at 9 months of age with vomiting, diarrhea, and poor weight gain. Results of stool examinations, abdominal ultrasound, and upper GI series were normal. The mother kept a daily log of foods that had precipitated the vomiting and diarrhea, with no clear pattern noted. The mother was described by the referring physician as a caring and involved parent. A 3 year old sibling was in good health.

Prior to evaluation, the child had demonstrated only a 500 gram weight gain over the prior three months. Upper GI endoscopy revealed a normal esophagus, stomach, and duodenum. A trial period wherein the child received only elemental formula failed to alleviate the vomiting or provide a substantial weight gain.

The vomiting and diarrhea continued with the child being hospitalized multiple times at different regional hospitals for dehydration. Premature ventricular contractions were noted. In addition, generalized muscle weakness developed, resulting in loss of ability to sit, stand, or walk.

Physical examination revealed a thin child with weight at the 5th percentile, height at the 75th percentile. He had marked muscle weakness, more prominent proximally. Head control was poor, and he could not stand or sit without support. The remainder of the neurologic examination was normal. Multiple PVCs were noted as well.

Abnormal laboratory values included a CPK of 3,507. EKG revealed PACs and PVCs. An echocardiogram was normal. EMG and nerve conduction studies were normal. Ipecac poisoning was suspected and confirmed by the presence of emetine in the serum and urine. The mother was unable to provide any explanation.

The child was placed in foster care, whereupon the vomiting ceased and weight gain returned to normal. His motor strength gradually returned and his CPK returned to normal.

Continued on back page
TOX TRIVIA:

1. What was the recent toxin that caused an epidemic of renal failure in Haiti by being a contaminant in liquid acetaminophen?

2. The "mad hatter" was poisoned by?

3. When was the first Poison Control Center established?

4. What was the toxin responsible for "St. Anthony's Fire"?

CLINICAL TOXICITY PEARLS:

1. Digoxin exposed patients may have depressed ST segments on ECG known as Salvador Dahlia's Mustache.

2. TCA exposed patients may have a rightward axis deviation of the terminal 40 msec of the QRS complex on ECG.

3. Neither of the above infer toxicity, only exposure.

NEWS BULLETINS:

Congratulations to our newly Certified Poison Information Specialists!!!

HISTORICAL MATCHING TRIVIA:

ANSWERS:

1. Ben Vincke
2. Amy Tan
3. Yvonne Lincoln
4. Elizabeth SX
5. George C.
6. Toby

REFERENCE:


Contributed by R. Cantor, M.D.