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Treatment Strategy for Hyponatremia

ABSTRACT & COMMENTARY

Source: Ayus JC, Arieff AI. Chronic hyponatremic encephalopathy in postmenopausal women. *JAMA* 1999;281:2299-2304.

In a nonrandomized, prospective study, 53 encephalopathic postmenopausal women with symptomatic, chronic hyponatremia (Na^+ < 130 mmol/L) were treated in one of three ways: intravenous sodium chloride before respiratory insufficiency (n = 17); intravenous sodium chloride after respiratory insufficiency (n = 22); or fluid restriction only (n = 14). Treatment approach was determined by when the authors were consulted to guide therapy. They were directly involved in the treatment of the first group; whereas in the other two groups, they were not involved until the patients had developed respiratory insufficiency (defined as either respiratory arrest or pO_2 < 50 mmHg). Chronicity of hyponatremia was defined as either a documented duration of 48 hours or longer or a rate of decrease of less than 0.5 mmol/L per hour over at least 48 hours. Intravenous sodium chloride therapy involved both "normal" and hypertonic saline.

Neurological outcome, initially and at follow-up, did not correlate with initial plasma sodium concentration or rate of correction of the hyponatremic state. The neurologic outcome was dramatically better in the first group, as was the mortality data. All patients without hypoxia experienced full recovery. The authors conclude that chronic, symptomatic hyponatremia in postmenopausal women is not a benign entity, and that intravenous sodium chloride therapy is both safe and effective.

COMMENT BY RICHARD HARRIGAN, MD, FAAEM, FACEP

The treatment of serious hyponatremia has been a subject of debate in the literature, as researchers try to determine predictors of morbidity and mortality and balance the benefits of aggressive therapy with the risks, most notably myelinolysis (formerly central pontine myelinolysis).[1] Acute hyponatremia has been regarded as more malignant than chronic, although this has been questioned,[2] and the current study certainly casts some doubt on this

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INSIDE

position. As the accompanying editorial highlights, hypoxia seems to be operative in the determination of morbidity and mortality in these patients.[3] It seems to be important to be aggressive with these patients—if they have mental status changes attributable to the low sodium—regardless of the acuity/chronicity of the hyponatremic state. Although chronic hyponatremia is a risk factor for the development of myelinolysis,[1] the rate of correction is probably more important—overly rapid correction leads to an increased likelihood of myelinolysis.[1-3] It is also important to note that none of the patients in this study had "hypervolemic hyponatremia," wherein the low sodium is attributable to underlying cirrhosis, nephrosis, or heart failure; these patients had either hypovolemic or euvolemic hyponatremia. ❖

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2. Votey SR, et al. Disorders of water metabolism: Hyponatremia and hypernatremia. *Emerg Med Clin North Am* 1989;7:749-769.
3. Knochel JP. Hypoxia is the cause of brain damage in hyponatremia (editorial). *JAMA* 1999;281:2342-2343.

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Unsuspected Pneumothorax on Abdominal CT: Is a Chest Tube Needed?

ABSTRACT & COMMENTARY

Source: Brasel K, et al. Treatment of occult pneumothoraces from blunt trauma. *J Trauma Inj Infec Crit Care* 1999;46:987-991.

Adult blunt trauma victims admitted to either of two institutions were prospectively randomized to either tube thoracostomy or observation for occult pneumothorax (PTX), defined as PTX seen on abdominal CT scan but not on AP chest radiograph as interpreted by the trauma chief resident or attending. Positive pressure ventilation was not an exclusion criterion. Although 86 patients with 98 occult PTXs were eligible, only 39 patients with 44 PTXs were enrolled. This was attributed to a variety of reasons, including physician judgment, refusal to give informed consent, and delay in diagnosis; in roughly one-third of the unenrolled patients, the reason for exclusion could not be determined.

Respiratory distress and PTX progression were the main outcome measures. There was no significant difference between groups in either of these two parameters. The three patients (14%) that did develop respiratory distress in the observation group did so for reasons unrelated to their PTX. Three patients in the observation group developed progression of PTX; two were on positive pressure ventilation and received chest tubes, while the other was observed and did well. Across groups (treated, observed, and unenrolled), PTXs that progressed tended to be larger (defined, as in previous literature, as 5×80 mm), although this did not reach statistical significance. The authors conclude that these occult PTXs can be cautiously observed without tube thoracostomy, even if the patient will be on a ventilator.

COMMENT BY RICHARD HARRIGAN, MD, FAAEM, FACEP

As the authors acknowledge in their discussion, this study is at odds with previous literature, both prospective and retrospective, regarding the need for a chest tube for occult PTX in patients that will be receiving positive pressure ventilation. As the discussants mention in the feature that follows the paper, this study has the methodologic concern of selection bias, as more than half of the eligible patients were not enrolled. Moreover, the incidence of complications, which were the main outcome measure, was small. Further study is probably needed

before this study is embraced fully, particularly since, in an earlier study, eight of 15 patients on positive pressure ventilation randomized to the observation arm developed progression of their PTXs, and two developed tension PTX.[1] Possible explanations offered for these discrepant findings included changes in ventilator management philosophy, with a more recent trend toward lower tidal volumes and lower peak airway pressure limits. ❖

Reference

1. Enderson BL, et al. Tube thoracostomy for occult pneumothorax: A prospective randomized study of its use. *J Trauma* 1993;35:726-730.

Screening for Serious Bacterial Illness in Febrile Neonates

ABSTRACT & COMMENTARY

Source: Baker MD, Bell LM. Unpredictability of serious bacterial illness in febrile infants from birth to 1 month of age. *Arch Pediatr Adolesc Med* 1999;153:508-511.

Reliable screening tools have been developed and validated to identify selected groups of febrile infants 1-2 months of age who are at low risk for serious bacterial illness (and therefore safe to manage as outpatients). One commonly applied screening tool is the "Philadelphia Protocol." The Philadelphia Protocol identifies the low risk 1-2-month-old baby as having a well appearance; no obvious source of bacterial infection on physical exam; WBC less than 15 K; band/neutrophil ratio less than 0.2; UA with less than 10 cells/hpf and no bacteria; CSF with less than 8 cells and normal gram stain; a normal CXR; and no WBC or blood in stool.

Can the Philadelphia Protocol accurately identify febrile babies younger than 1 month of age who are at low risk of serious bacterial infection? The authors of the Philadelphia Protocol report on applying it to this population. Over a recent 36-month period, 254 febrile neonates 3-28 days of age who were admitted to the Children's Hospital of Philadelphia were enrolled in the current study. All of these neonates were treated with empiric antibiotics pending culture results. The study investigators reviewed the medical records, applied the Philadelphia Protocol, and retrospectively judged its safety and efficacy. The incidence of serious bacterial illness in the 254 neonates was 12.6% (30 of 254). The bacterial diseases diagnosed were: 17 urinary tract infections, eight cases of bacteremia, four cases of meningitis, two cases of gastroenteritis, and five other.

When the Philadelphia Protocol was applied to the 254 febrile neonates, 109 met the criteria for "low risk" for serious bacterial disease. Of these 109, five were diagnosed with serious bacterial disease (2 with urinary tract infection, 2 with meningitis, and 1 with gastroenteritis). The performance of the Philadelphia Protocol as a screening tool to identify serious bacterial illness in this population shows a sensitivity of 84% (5 of 32) with a 95% confidence interval of 67-95%, and a negative predictive value of 95% (104 of 109) with a 95% confidence interval of 90-99%.

COMMENT BY LEONARD FRIEDLAND, MD

The Philadelphia Protocol cannot safely identify febrile neonates at low risk for serious bacterial illness (and, therefore, safe to manage as outpatients). Applying this screening tool would have missed five patients (with 95% confidence up to 10% of patients) with serious bacterial illness. These data support that the initial management of the febrile baby younger than 1 month of age should include a complete evaluation for serious bacterial illness, hospitalization, and the administration of empiric antibiotics. ❖

TEE in the ED—What are its Complications?

ABSTRACT & COMMENTARY

Source: Gendreau MA, et al. Complications of transesophageal echocardiography in the ED. *Am J Emerg Med* 1999;17:248-251.

Gendreau and colleagues, in a retrospective review of 184 consecutive transesophageal echocardiography (TEE) procedures performed in an urban level I trauma center and tertiary care hospital ED, sought to determine the complication rate and high-risk patient subsets in ED patients undergoing this procedure. Prior to chart review, definitions for major and minor complications were established. Forty patients were excluded since TEE was performed outside of the ED; two others were excluded due to inadequate chart documentation. Of the remaining 142 patients, 88 were victims of trauma and underwent TEE to rule out thoracic aortic injury, 53 underwent TEE to rule out aortic dissection, and one patient underwent TEE to rule out prosthetic aortic valve thrombosis. Eighteen (12.6%) patients experienced complications of ED TEE— eight (5.6%) were major and 10 (7%) were minor complications. *(See Table.)* Patient variables (age, gender, use of sedation, hemodynamics,

oxygen saturation, hematocrit, and serum bicarbonate) failed to yield subsets predictive of complications. The authors conclude that, compared to literature reporting a complication rate of 0-5% for TEE performed in the non-ED setting,[1-2] TEE performed in the ED carries a higher complication rate, and that no pre-procedural parameters are predictive of complications.

Table

Complications of TEE in the ED

	Trauma (n = 88)	Medical (n = 54)
Major		
Death	0	1
Respiratory insufficiency/ failure	4	3
Minor		
Transient hypotension	1	2
Minor dysrhythmia	1	0
Emesis without aspiration	4	0
Agitation	1	2

COMMENT BY FREDERIC H. KAUFFMAN, MD, FACEP

As with all diagnostic techniques dependent upon operator expertise, as more experience is gained with TEE in the ED setting, the procedure will yield increasingly greater degrees of accurate information with lesser "clinical cost" to the patient. One must always be cautious, however, in evaluating preliminary reports touting the safety and efficacy of new clinical tools. The authors, appropriately, have raised a cautionary flag regarding the use of TEE in the ED setting.

There are several explanations for the high ED TEE complication rate seen in this study. First, data collection began eight years ago. TEE efficacy and safety have improved since its expanded use began in the early 1980s, and certainly during the past decade. Second, not all prior studies evaluating the role of TEE looked at complication rate as a primary study end point. A commonly referenced article by Smith and colleagues found TEE to be accurate and safe in the evaluation of aortic rupture in trauma patients.[1] There were no reported complications of the procedure, but eight patients were excluded from evaluation due to inadequate sedation. Such patients would have been considered by Gendreau and colleagues to have experienced complications of TEE. Third, it makes intuitive sense that nearly any major procedure performed in the ED would carry a greater complication rate compared to its performance outside the ED. ED patients are, by their very nature, "emergent." Some procedures are performed in the ED on patients who ultimately die in the ED or shortly after transfer to another unit. Waiting for full stabilization may not be appropriate, but such patients would never be part of a non-ED study of TEE. Lastly, quite appropriately, the authors highlight the fact that respiratory insufficiency/failure was the most common complication of ED TEE. Airway management always assumes priority over all other issues, and though no cause and effect conclusion can be drawn from this study, ED airway management must be aggressive with not just a reactionary, but also an anticipatory, approach to complications. ❖

Reference

1. Smith MD, et al. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med* 1995;332:356-362.
2. Pearson AC, et al. Safety and utility of transesophageal echocardiography in the critically ill patient. *Am Heart J* 1990;119:1083-1089.

Abciximab for Unstable Angina in Patients with Elevated Troponin T

ABSTRACT & COMMENTARY

Source: Hamm CW, et al. Benefit of abciximab in patients with refractory unstable angina in relation to serum troponin T levels. *N Engl J Med* 1999;340:1623-1629.

This study is an important subgroup analysis from the c7E3 Fab Antiplatelet Therapy in Unstable Refactory Angina (CAPTURE) trial. In the earlier study, the glycoprotein IIb/IIIa-receptor blocker abciximab (ReoPro) was shown to reduce the risk of myocardial infarction (MI) in patients with refractory unstable angina.[1] This study looks more closely at the prognostic value of troponin T and the effect of abciximab on risk of adverse outcome in relation to troponin T level. The patients had recurrent chest pain at rest associated with ECG changes consistent with ischemia and continued to have chest pain despite treatment with IV heparin and nitroglycerin. All patients had significant coronary artery disease documented by angiography. Patients were excluded if they had sustained an MI within the prior two weeks.

Suitable patients were given abciximab 0.25 mg/kg IV bolus followed by a 10 mcg/min infusion or a matching placebo. Subjects were scheduled for percutaneous transluminal coronary angioplasty (PTCA) the day after study medication was given. Study end points were death, MI,

unscheduled PTCA, and coronary bypass surgery.

Of the 1265 patients in the CAPTURE trial, 890 met criteria for inclusion in this study. Subjects were grouped according to troponin T levels at study entry. Among the 615 patients with normal troponin T levels, the risk of death or MI was 8% and was not improved by abciximab treatment. However, the effect of abciximab was striking among the 275 patients with elevated troponin T levels (> 0.1 ng/mL): 7.5% of abciximab-treated patients experienced death or MI within six months, compared to 24% of placebo-treated patients. The relative risk of death or MI associated with abciximab treatment was 0.32 in patients with elevated troponin T levels. The authors conclude that troponin T elevation identifies a subgroup of patients at high risk of death or MI and that this group will benefit considerably from treatment with abciximab.

COMMENT BY DAVID J. KARRAS, MD, FAAEM, FACEP

Unstable angina carries with it a high risk of MI and/or death within several months of onset. The rate of adverse events in placebo-treated patients (despite therapy with heparin and nitroglycerin) in this study is strikingly high and consistent with earlier studies. Furthermore, this study confirms that troponin T elevation greatly increases the risk of mortality and infarction. Troponin T is an extremely sensitive and specific marker of myocardial injury. Elevated troponin T levels in patients without a rise in creatine kinase is thought to reflect focal myocyte necrosis from embolization of thrombi released from a disrupted coronary plaque. Since such plaque disruption precedes coronary occlusion, troponin T elevation often heralds an impending major infarction or sudden cardiac death.

Certain caveats must be condidered before applying this study's finding to patients in the ED. Most importantly, abciximab was not used as primary therapy for unstable angina. The patients in this study all had recurrent chest pain despite management with heparin and nitroglycerin. All subjects were demonstrated to have significant coronary artery disease; the results may not be applicable to "all comers" presenting with angina-like chest pain to the ED. Nonetheless, it is important that emergency physicians be familiar with the glycoprotein IIb/IIIa-receptor blockers. Several articles discussing the role of these drugs from an ED perspective appear in a recent issue of *Journal of Emergency Medicine* (1999;17:565-595). ❖

Reference

1. The CAPTURE investigators. Randomized placebo-controlled trial of abciximab before and during coronary intervention in refractory unstable angina. *Lancet* 1997;349:1429-1435.

Special Feature

Postpartum Toxemia

By William Brady, MD

Preeclampsia is characterized by hypertension, edema, and proteinuria and frequently occurs in nulliparous women. The syndrome, noted in a minority of pregnancies, usually becomes apparent after the 20th week of pregnancy, most often near the end of the third trimester. A variant of the syndrome (the HELLP syndrome) includes the above clinical findings as well as hemolytic anemia, thrombocytopenia, coagulopathy, and elevations in liver enzymes. The acronym "HELLP" describes the characteristics of the syndrome: "H" for hemolysis, "EL" for elevated liver enzymes, and "LP" for low platelets. Preeclampsia is noted less commonly in patients during the first half of pregnancy, and is usually associated with molar or hydropic degeneration of the placenta. With the appearance of generalized seizure activity, preeclampsia has progressed to eclampsia, or toxemia of pregnancy. Eclampsia complicates less than 1% of deliveries and constitutes a significant life threat to both mother and fetus. Maternal mortality is reported to approach 15% and perinatal mortality ranges from 10% to 30%. [1-4] Toxemia occurs most often antepartum; however, a significant minority of cases develop in the postpartum period. Postpartum toxemia may have its onset only moments after delivery is complete or, alternatively, the disorder may manifest as late as three weeks after delivery. Emergency physicians must be familiar with the presentation and management of toxemia. Further, they must recognize that a significant minority of toxemic patients will experience the onset of eclampsia following uncomplicated delivery and discharge from the hospital.

Epidemiology

An analysis of the incidence of toxemia relative to the timing of pregnancy reveals that the majority of patients experience the onset of seizure activity antepartum. In general, 80% of toxemic patients will have the first seizure in the ante- or intrapartum periods. A review of past eclampsia studies demonstrates that approximately 20% of patients will convulse initially in the postpartum period.[5] The past several decades have demonstrated a decline in the occurrence of eclampsia, most likely reflecting not only increased recognition of preeclampsia, but also improved means of treating such patients, perhaps preventing the progression to toxemia. During the same time period, a relative increase in the propor-

tion of postpartum cases has been noted.

Significant controversy exists in the obstetrical community, however, concerning the definition of postpartum toxemia in terms of the time of onset of the convulsions relative to delivery. One perspective maintains that the vast majority of such cases will occur in the first 24 hours postpartum, with the remainder taking place during the second day after delivery. Further, seizures first appearing beyond 48 hours postpartum, theorized to result from eclampsia, are regarded with skepticism by this group. Others have placed less emphasis on the time of onset of the first seizure and have adopted the term "late postpartum eclampsia" to describe patients who develop toxemia more than 48 hours after delivery of the infant. Regardless of one's perspective, the emergency physician should consider the following issues in the approach to such a patient: 1) a significant number of toxemic patients will first manifest the syndrome more than 48 hours after delivery; 2) a thorough medical evaluation is warranted in all such patients to rule out other causes of convulsion; and 3) that, while excluding other causes of seizures, such patients should be aggressively treated for presumed postpartum eclampsia.

Pathophysiology and presentation

The pathophysiology of preeclampsia and eclampsia remains largely unknown, yet it is clear that the pathogenesis involves the uteroplacental bed. The pathophysiologic end point is severe vasospasm with resultant hypertension. It is speculated that this vasospasm may be secondary to increased vasoactive substances. The concentration of the vasodilating prostaglandin, prostacyclin, is lower in women with preeclampsia and eclampsia, likely causing vasospasm due to unopposed action of the vasoconstricting agent angiotensin II. The seizure activity in eclampsia is a direct manifestation of a failure of the cerebral autoregulatory mechanisms in the control of intracranial perfusion at elevated blood pressures. Such a pathophysiologic failure supports the clinically proven theory that the best anticonvulsant therapy is proper management of hypertension.

The clinical presentation of eclampsia in the postpartum period is not markedly different from the antepartum version of toxemia, with the obvious exception of the lack of a fetus. Patients may present in the postpartum period with complaints suggestive of impending convulsion including headache, visual changes, and epigastric pain of several days duration. Patient age and parity also are similar in both antepartum and postpartum toxemia; young, nulliparous patients are most frequently affected. Physical examination and laboratory findings such as edema, hyper-reflexia, hypertension, and proteinuria are noted at similar rates. Hypertension and hyper-reflexia are found in the majority of cases, while edema and proteinuria are present in approximately 50% of patients. A previous diagnosis of preeclampsia at the time of convulsion is available in 50-75% of patients with postpartum eclampsia. The remaining patients represent cases in which the diagnosis was missed or the process appeared suddenly with rapid progression in the postpartum period. The severity of eclampsia appears to differ between the ante- and postpartum varieties. Postpartum toxemia is described as mild in two-thirds of cases with both fewer than five seizure episodes and less pronounced elevations in blood pressure in the majority of patients. Conversely, antepartum eclampsia is characterized as severe in approximately 60% of cases, with the majority of patients experiencing greater than 10 convulsive episodes. Further, postpartum patients receiving adequate therapy are more likely to respond favorably in terms of suppression of seizure activity and control of blood pressure when compared with antepartum toxemic cases.[6]

Evaluation and management

The evaluation of postpartum patients presenting with findings potentially consistent with eclampsia must include a thorough physical examination with special emphasis on the nervous system. A metabolic panel including electrolytes and glucose is essential. A rapid, bedside determination of the serum glucose is mandatory to rule out hypoglycemia as a cause of the seizure. Toxicologic studies for epileptigenic agents such as cocaine or amphetamine must be considered in appropriate cases. The urine must be examined for the presence of protein. The complete blood count may reveal hemoconcentration as well as thrombocytopenia; coagulation studies may demonstrate prolongation of the prothrombin and partial thromboplastin times. The liver function tests, specifically the transaminases, are frequently elevated, especially in the HELLP syndrome variant. A CT scan of the head should also be performed at the time of presentation to the ED. Analysis of the cerebrospinal fluid may be warranted depending on the clinical presentation. The above evaluation is performed to rule out other causes of seizure in the postpartum patient. The differential diagnosis for such convulsions includes metabolic, toxic, infectious, neurologic, and traumatic etiologies, as well as pre-existing or new-onset epilepsy.

The initial management of postpartum eclampsia is similar to that of antepartum toxemia. Attention to the ABC's with maintenance of an airway, satisfactory ventilation and oxygenation, and adequate perfusion is

essential. For patients presenting with active seizure, a parenteral benzodiazepine such as lorazepam or diazepam followed by intravenous phenytoin or fosphenytoin at conventional doses is a reasonable approach. If the diagnosis of postpartum eclampsia is suspected, treatment with intravenous magnesium sulfate with its anticonvulsant and antihypertensive properties is suggested, though not universally accepted among the obstetrical and neurologic communities. It is administered in a 4-6 g intravenous bolus over 30 minutes followed by a constant infusion of 1-2 g/h. Manifestations of excessive magnesium therapy include lethargy, loss of reflexes, hypotension, and, ultimately, respiratory depression. The continuous infusion should be stopped if reflexes disappear or the serum magnesium level exceeds 8 mEq/L. Parenteral calcium may be used to treat magnesium toxicity if profound. Hypertension may be further managed with hydralazine (the traditional agent) or other medications (such as nifedipine, labetolol, or nitroprusside) titrated to the clinical picture. Diuretic agents are not helpful and may actually worsen the situation by impairing already compromised perfusion. In general, the emergency physician has more flexibility in the choice of medications in the management of postpartum eclampsia as compared with antepartum toxemia, due to the absence of the fetus. Patients presenting with toxemia or a constellation of clinical findings suggestive of toxemia in the postpartum period should be admitted to a monitored setting with both urgent obstetrical and neurologic consultations. Such patients presenting with complaints and/or physical findings consistent with impending seizure or in the postictal state should be aggressively treated with magnesium sulfate as well as antihypertensive agents, with the aim of halting disease progression. ❖

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5. Brady WJ. et al. Postpartum toxemia: Hypertension, edema, proteinuria, and unresponsiveness in an unknown female. *J Emerg Med* 1995;13:643-648.
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CME Questions

14. Abciximab reduces the risk of MI and death associated with unstable angina when administered:

a. upon patients' ED presentation.
b. regardless of serum troponin T level.
c. to patients with or without known atherosclerotic disease.
d. concomitantly with heparin.

15. In young infants with fever, all of the following are correct *except*:

a. reliable screening tools have been developed and validated to identify selected groups of febrile babies 1-2 months of age who are at low risk for serious bacterial illness.
b. reliable screening tools have been developed and validated to identify selected groups of febrile babies younger than 1 month of age who are at low risk for serious bacterial illness.
c. urinary tract infections account for the largest proportion of serious bacterial illness in the febrile neonate.

16. TEE in the ED:

a. may have greater complications than in non-ED settings due to the emergent nature of the patients being evaluated.
b. is safe in medical, but not trauma, patients.
c. does not cause complications if patients undergo prior intubation.
d. is better than aortography in diagnosing aortic rupture.

17. Postpartum toxemia:

a. is more common than the antepartum variant.
b. is largely attributed to prenatal cocaine abuse.
c. is more likely to have a mild than a severe course.
d. is attributable to postnatal cocaine abuse.

18. Reasonable pharmacologic adjuncts in the treatment of postpartum toxemia include all of the following *except*:

a. phenytoin.
b. lorazepam.
c. magnesium sulfate.
d. calcium gluconate.

19. When treating an elderly, dehydrated woman on thiazides in the ED with a sodium of 110 mEq/L and obtundation, it is probably reasonable to:

a. recommend her for emergent hemodialysis to correct the sodium.
b. administer intravenous sodium chloride at a controlled rate and admit her.
c. admit her with fluid restriction if the hyponatremia is chronic.
d. perform a head CT and continue the thiazide if there is no evidence of cerebral edema.

20. In the study by Brasel et al on the need for chest tube with occult pneumothorax (PTX), which of the following statements is true?

a. All patients on positive pressure ventilation eventually needed a chest tube.
b. Occult PTXs secondary to penetrating trauma did better than those due to blunt trauma.
c. Occult PTX was defined as PTX not seen on the initial plain chest film or CT scan of the abdomen.
d. Observation of occult PTX was not associated with an increased risk of PTX progression or respiratory distress, but the incidence of these complications was low.

Peaked T Wave Abnormalities

By Ken Grauer, MD

Clinical Scenario: The ECG in the figure was obtained from a 50-year-old man who presented to the office for evaluation of atypical chest "tightness" over the preceding few months. In view of the fact that the serum potassium was normal at the time this ECG was recorded, *how would you interpret this tracing?*

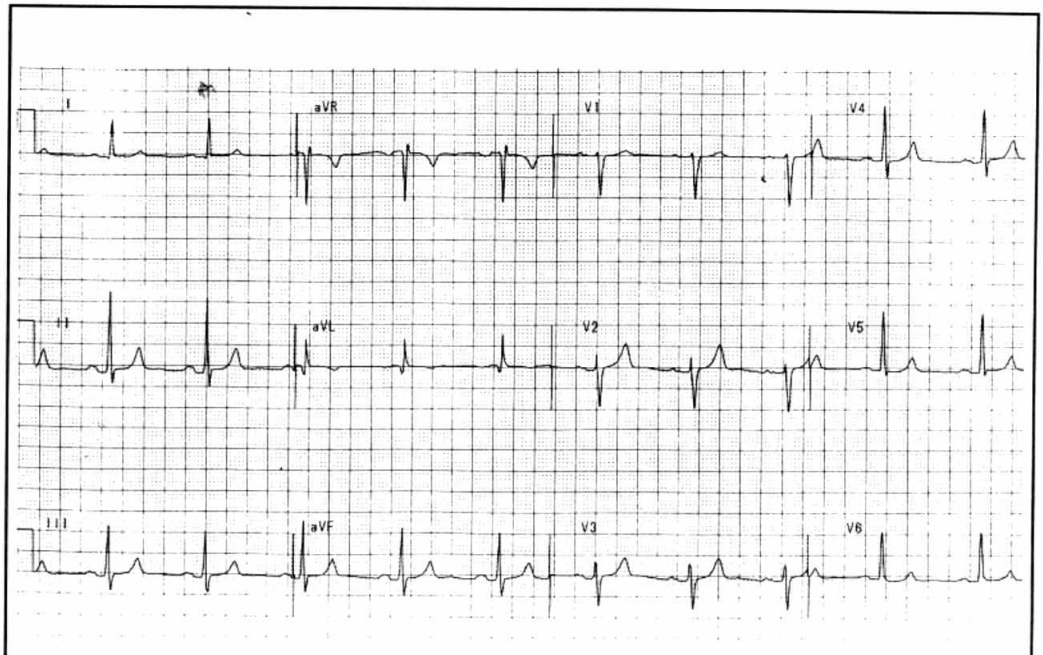

Figure. ECG recorded on a 50-year-old man with chest "tightness." Serum potassium is normal.

Interpretation: The rhythm is sinus at a rate of about 65 beats/min. All intervals are normal. The mean QRS axis is approximately +45°. There is no evidence of chamber enlargement. The finding of note on this tracing relates to assessment of ST segments and T wave appearance. Specifically, ST segments are flattened in many leads and T waves are peaked. The point to emphasize is that this tracing should *not* be interpreted as a normal ECG. Admittedly, the abnormalities are subtle—yet they are definitely present.

Normally, ST segments in most leads manifest a slightly rounded and upward sloping concavity, blending almost imperceptibly into an upright T wave. This is not the case in the figure. Instead, there is *straightening* of ST segments—especially in leads II, III, aVF, V_3, V_4, and V_5. This subtle finding may be a *nonspecific* indicator of underlying coronary disease. Unfortunately, many other entities also produce a similar ECG appearance (ergo designation of ST segment flattening as a "nonspecific" change).

T wave peaking is seen in virtually the same leads on this tracing that show ST segment flattening. The presence of T wave peaking should always suggest the possibility of hyperkalemia. However, serum potassium is normal in this case. T wave peaking is also commonly seen as a normal repolarization variant in otherwise healthy individuals. Distinction from the T wave of hyperkalemia is usually suggested by history (that the patient is healthy and asymptomatic) and the findings that with a repolarization variant the peak of the T wave tends to be rounded, the ascending and descending limbs of the T wave are not as symmetric, and the base of the T wave is wider. A serum potassium level should always be checked if the diagnosis is in doubt.

Not nearly as well appreciated is the fact that T wave peaking may sometimes reflect ischemia. None of the standard leads on a 12-lead tracing directly view the posterior wall of the left ventricle. Posterior wall involvement must, therefore, be viewed *indirectly* on the ECG—by assessing for changes that occur in *anterior* leads (i.e., leads V_1, V_2, and V_3) that reflect a "mirror image" view of electrical events in the posterior wall. Instead of the usual manifestation of ischemia (i.e., deep symmetric T wave inversion), posterior ischemia may, therefore, produce symmetric T wave peaking.

Thus, in this patient with a history of chest discomfort, the finding of ST segment flattening in many leads in conjunction with T wave peaking should suggest the possibility of ischemia. The presence of coronary disease was confirmed with further testing. ❖

In Future Issues: **Selected papers from the 1999 national SAEM meetings**