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Non-Operative Management for Abdominal Gunshot Wounds?

ABSTRACT & COMMENTARY

Source: Velmahos GC, et al. Selective nonoperative management of 1856 patients with abdominal gunshot wounds: Should routine laparotomy still be the standard of care? *Ann Surg* 2001;234:396.

The authors performed a retrospective chart review of 1856 patients with abdominal gunshot wounds (1405 anterior, 451 posterior) admitted during an eight-year period at Los Angeles County Medical Center. According to a previously developed protocol, these patients underwent either laparotomy or selective nonoperative management (SNOM). Patients who did not have peritonitis, were hemodynamically stable, and had a reliable clinical examination (absence of head injury, intoxication, spinal cord injury) were observed. Patients in the SNOM group underwent CT scanning to define bullet trajectory and organ injury. Frequent serial examinations on individual patients were performed by the same treating surgeon, typically a junior resident, for a 24-hour period. Patients who experienced a change in their clinical status underwent delayed laparotomy.

Initially 792 (42%) patients (34% of patients with anterior and 68% of patients with posterior abdominal gunshot wounds) were selected for nonoperative management. During observation, 80 patients (4% of total; 10% of SNOM group) developed symptoms and required delayed laparotomies, which revealed organ injury requiring repair in 57. The majority of delayed laparotomies occurred within eight hours of presentation. Five patients (0.3% of total; 0.6% of SNOM group) suffered complications potentially related to delay in laparotomy. Four of these five were intraabdominal abscess; all were managed successfully. Seven hundred-twelve patients (38%) were managed successfully without operation. The rate of non-therapeutic laparotomy was 14%. Compared with patients with unnecessary laparotomy, patients managed without surgery had significantly shorter hospital stays and lower hospital charges.

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INSIDE

Readers:

Please see enclosed insert for details on our Nov. 14 audio conference, "Responding to the Unimaginable: How Saint Vincents Coped with the World Trade Center Attack."

COMMENT BY MICHAEL A. GIBBS, MD, FACEP

Classic mantra underscores the importance of immediate laparatomy in all patients with abdominal gunshot wounds. This assertion is based on the long-held belief that the rate of intra-abdominal organ injury in this population approaches 90%. While this may be true for military wounds, more recent data suggest that abdominal gunshot wounds from civilian violence are associated with a much lower incidence of clinically significant intra-abdominal injuries, ranging from 30-74%.

This study confirms the results of two smaller prospective studies by the same authors, demonstrating that roughly one-third of anterior abdominal gunshot wounds[1] and two-thirds of posterior gunshot wounds[2] can be managed without surgery.

I think this approach undoubtedly will become more popular at major trauma centers with experienced trauma surgeons and dedicated resources. It is unlikely ever to become reality at most small centers. After all, few hospitals can boast the Los Angeles experience of 232 abdominal gunshot wounds per year! While this study has several important weaknesses (most importantly a lack of precise definitions for the indications for surgery), it represents an important paradigm shift that challenges traditional surgical dogma. ❖

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Trichomoniasis in Pregnancy: To Treat or Not to Treat?

ABSTRACT & COMMENTARY

Source: Klebanoff MA, et al. Failure of metronidazole to prevent preterm delivery among pregnant women with asymptomatic trichomonas vaginalis infection. *N Engl J Med* 2001; 345:487-493.

This large, randomized, double-blinded trial of metronidazole vs. placebo in pregnant women with asymptomatic trichomoniasis was conducted to assess whether therapy would reduce the risk of preterm delivery. Asymptomatic pregnant women between 8 and 23 weeks gestation were screened at 15 sites for *Trichomonas vaginalis* by culture of vaginal secretions. After exclusions were accounted for, 617 women with trichomoniasis who were 16-23 weeks gestation were randomized to receive two 2 g doses of metronidazole (320 women) or placebo (297 women) 48 hours apart. The women were treated a second time with the same two-dose regimen at 24-29 weeks gestation. The primary outcome was delivery before 37 weeks of gestation. The two groups were similar in terms of numerous characteristics measured at randomization. Approximately 90% were black, about 76% never married, and the average age was 22 years. Both groups were similar in their compliance with the medication, but side effects were significantly more common in the metronidazole group.

Delivery occurred before 37 weeks gestation in 19% of women in the metronidazole group and in 10.7% in

the placebo group (relative risk, 1.8; 95%, CI 1.2-2.7; P = 0.004). Most of the difference was due to an increase in the rate of spontaneous preterm labor in the metronidazole group (10.2% vs 3.5%; relative risk, 3.0; 95% CI, 1.5-5.9). In the 529 women who had follow-up cultures, *T. vaginalis* persisted in 65% of women who received placebo and 7% of women who received metronidazole.

COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

As with many studies, this large, well-designed, randomized trial raised as many questions as it answered. The significant finding was that, despite the fact that infection with *T. vaginalis* has been associated with an increase in adverse outcomes in pregnancy, treatment with metronidazole did not prevent preterm delivery. In fact, there was an increase in the rate of spontaneous preterm labor in the metronidazole group, and this unanticipated result led to the cessation of the study.

The results of this study are consistent with results from the same researchers in a previous trial of metronidazole therapy for bacterial vaginosis (BV).[1] The BV study found that treating pregnant women for asymptomatic BV with metronidazole did not reduce the risk of preterm birth, despite its effectiveness in eliminating BV. However, preterm delivery was more frequent only in a subgroup of women with both BV and trichomonas who received metronidazole. An increase in preterm delivery also has been observed in pregnant women with BV who were treated with clindamycin cream.[2] In still another trial, a regimen of metronidazole and erythromycin increased the occurrence of preterm delivery among women who did not have BV, but reduced it in those who had BV.[3] As a possible explanation for these findings, the authors suggest that there may be a group of women in whom the risk of adverse outcomes of pregnancy somehow is increased by antibiotic treatment, although the reasons for this are unclear. In addition, it seems doubtful that the association of metronidazole treatment with an increased risk of preterm delivery was due to the metronidazole itself. If this were the case, the effect most likely would have been seen earlier, instead of at 35-36 weeks gestation, when the outcome differences became significant.

In the emergency department we now are confronted with a clinical dilemma. Do we treat pregnant patients who have trichomoniasis? The easy answer is that if they are asymptomatic, probably not. There does not seem to be any reason to look routinely at wet mounts in pregnant women without vaginal symptoms.

However, if a pregnant woman is symptomatic, the answer is less clear. This study excluded women with vaginal symptoms, who presumably were treated but not reported on. Until there is more research to guide us, we will be weighing the risks and benefits on a case-by-case basis or deferring the decision to the patient's obstetrician in a follow-up visit. After discussing this study, several OB-GYN colleagues said they still would consider treatment for women who are symptomatic. ❖

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Pneumococcal Infection and Antibiotic Therapy

ABSTRACT & COMMENTARY

Source: Schrag S, et al. Effect of short-course, high-dose amoxicillin therapy on resistant pneumococcal carriage. *JAMA* 2001;286:49-56.

Streptococcus pneumoniae bacteria commonly colonize the nasopharynx of asymptomatic children. However, invasive pneumococcal infections, such as otitis media (OM), sinusitis, meningitis, pneumonia, and bacteremia, result in significant morbidity and mortality worldwide. The growing prevalence of penicillin and multi-drug resistant strains of the pneumococcus have raised considerable alarm in the medical community.

Because antibiotic use and overuse has been cited as one of the chief causes of emerging resistance, some have suggested that changes in antibiotic prescribing practices may reduce the spread of resistant organisms. In this study, the authors sought to determine whether a short-course, high-dose antibiotic regimen would beneficially impact nasopharyngeal carriage rates of resistant pneumococcus in children being treated for respiratory tract infections.

The authors conducted a prospective, single-center trial in 795 children ages 6-59 months who required

antibiotic therapy for acute respiratory tract infections, OM, pneumonia, or sinusitis. Subjects were randomized to receive amoxicillin at a standard dose regimen (40 mg/kg/d divided bid) for 10 days, or a new high-dose, short-course regimen (90 mg/kg/d divided bid) for five days. Investigators measured pneumococcal nasopharyngeal carriage rates at days 0, five, 10, and 28 for both susceptible and drug-resistant strains. They also measured patient compliance with the regimens (defined as completing 80-120% of the medication by volume), as well as adverse reaction rates.

At baseline, pneumococcal carriage rates were 76% and 73% for the short-course/high-dose and standard regimen groups, respectively. As would be expected with antibiotic therapy, overall carriage rates declined on days five, 10, and 28 for both groups. Investigators found significantly lower rates of penicillin-resistant pneumoccoccus carriage with the short-course/high-dose therapy than the standard regimen (24% vs 32%, RR = 0.77, P = 0.03). There was a similar trend in resistance to trimethoprim-sulfamethoxasole (17% vs 23%, respectively, RR = 0.77, P = 0.08). Adherence to therapy was significantly better with the short-course/high-dose regimen (82% vs 74%, P = 0.02) and there was no difference in adverse event rates.

Based on their findings, the authors conclude that the short-course/high-dose amoxicillin therapy may be promising as an intervention to decrease the impact of antibiotic use on the emergence and spread of drug-resistant pneumococcus.

COMMENT BY THEODORE C. CHAN, MD, FACEP

This same short-course, high-dose amoxicillin therapy recently has been approved by the Food and Drug Administration. This regimen has been recommended by a number of groups for the treatment of acute OM in regions where pneumococcal drug resistance is high.[1] There are a number of theoretical advantages to this new regimen. First, the increased antibiotic concentrations from the higher dose can overcome penicillin resistance (which, unlike macrolide resistance, is not absolute). Second, the shorter course may reduce overall antibiotic exposure and selective pressure that can facilitate the emergence of resistant strains. While a number of studies suggest the shorter regimen has equal clinical efficacy, this is one of the first studies to suggest benefit in terms of reducing drug-resistance. This study found other benefits to the shorter therapy as well, including that patient compliance was improved with no increase in adverse effects.

It is important to note, however, that this study was performed at a single institution in the Dominican Republic. There may be important differences in both the pediatric population and pneumococcal strains in this developing country. For example, more than one-third of the pneumococcal strains at baseline were drug-resistant in this study. Recent work in the United States suggests the prevalence of drug-resistant pneumococcus approaches 25% but varies greatly by region.[2]

Finally, it is interesting to note that while both antibiotic regimens reduced overall pneumococcal carriage rates, the greatest impact was seen in drug-susceptible strains. In fact, for both regimens the actual ratio of drug-resistance to susceptible carriage increased by day 28, suggesting that despite therapy modifications, antibiotic use in general will continue to contribute to the emerging resistance problem. ❖

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Special Feature

Pharmacologic Treatment of Hypertensive Emergencies

By Maged Botros, MD, and David J. Karras, MD, FAAEM, FACEP

Hypertensive emergencies generally are defined as situations that require immediate blood pressure reduction to prevent or limit damage to target organs.[1] The decision to treat an elevated blood pressure as a hypertensive emergency is independent of the actual blood pressure value, but rather depends on evidence of acute end-organ damage. Examples of acute end-organ dysfunction related to hypertension include encephalopathy, intracranial hemorrhage, renal dysfunction, unstable angina pectoris, myocardial ischemia, acute left ventricular failure with pulmonary edema, aortic dissection, and eclampsia. Although the diagnosis of hypertensive emergency hinges on the presence of end-organ damage, most

authorities agree that it needs to occur in conjunction with a diastolic blood pressure greater than 110 mmHg.[2] The primary pathophysiologic abnormality in hypertensive emergencies is an alteration in autoregulation, in which important vascular beds are unable to constrict appropriately to maintain perfusion. Frank arteritis and ischemia usually follow this process.

The initial goal of therapy in hypertensive emergencies is a reduction in mean arterial pressure (MAP) of no more than 25% within the first minutes to two hours,[1] although the rate and degree of reduction vary somewhat depending upon the site of end-organ damage. Rapid drops in pressure should be avoided, as they may precipitate renal, cerebral, or coronary ischemia. Medical intensivists have traditionally preferred the use of sodium nitroprusside to accomplish this task; its rapidness of onset, short duration of action, and ease in titration have made it a convenient medication to use. However, given its extensive side effect profile, newer medications are being investigated to treat hypertensive emergencies with an emphasis on directly improving the function of the target end organ with evidence of damage.

Nitroprusside

Nitroprusside is a potent vasodilator that decreases both preload and afterload. Its onset of action is within seconds and duration of action is 1-2 minutes. Nitrocysteine is formed when nitroprusside reacts with the body's own cysteine and activates guanylate cyclase, which, in turn, stimulates the formation of c-GMP, a potent smooth muscle relaxant. Although nitroprusside achieves excellent blood pressure control, its side effect profile is of concern. In a large, randomized, placebo-controlled trial, nitroprusside was shown to increase mortality when infused in the early hours after a myocardial infarction (mortality at 13 weeks, 24.2% vs 12.7%).[3] This effect is believed to be due to a "cardiac steal" phenomenon, resulting in a significant reduction in cardiac blood flow in patients with significant coronary artery disease.

Furthermore, both clinical and experimental evidence demonstrate that nitroprusside increases intracranial pressure,[4-7] which may result in decreased cerebral blood flow. This phenomenon may have deleterious effects on patients presenting to the emergency department (ED) with hypertensive encephalopathy. Finally, cyanide is released nonenzymatically from nitroprusside in a dose-related manner. Cyanide is metabolized in the liver by thiosulfate-sulfurtransferase into the less toxic metabolite thiocyanate, which is excreted via the kidneys. Cyanide removal, therefore, requires adequate liver function, adequate kidney function, and adequate bioavailability of thiosulfate. The possibility of cyanide toxicity, therefore, must be considered in all patients receiving nitroprusside.

Considering the potential side effects, nitroprusside should be used with caution in patients presenting with renal, hepatic, or coronary artery impairment. Fortunately, a few therapeutic options have been developed and marketed in the past few years for the ED treatment of hypertensive emergencies.[8]

Fenoldopam

Fenoldopam is a selective DA_1 receptor agonist that has shown significant promise in the treatment of hypertensive emergencies, especially in patients exhibiting evidence of renal impairment. While fenoldopam is available in both oral and intravenous preparations, its serum half-life of only 10 minutes limits its utility as an oral agent. Dopamine acts at low doses by stimulating specific peripheral dopaminergic receptors that are classified into subgroups. DA_1 receptors are located post-synaptically on the smooth muscle of the renal, coronary, cerebral, and mesenteric arteries.[9] Stimulation of DA_1 receptors causes vasodilatation of those arteries. Intravenous fenoldopam is a potent DA_1 vasodilator that has 6-9 times the vasodilatory effect of dopamine. This vasodilatory effect tends to be strongest in the renal arteries, where renal blood flow can be increased by up to 77% in patients with impaired renal function.[10] In addition, recent evidence also suggests that DA_1 receptors also are located in the renal tubules, which, when stimulated, seem to be directly responsible for the natriuresis usually seen with dopamine administration.[10]

Although other antihypertensive agents produce a reduction in blood pressure by vasodilatory effects, the additional advantage of fenoldopam is its maintenance of renal perfusion combined with its natriuretic and diuretic effect. In addition, fenoldopam does not carry the added risk of cyanide toxicity seen in nitroprusside. In a prospective, randomized, multicenter, clinical trial, Panacek and colleagues compared fenoldopam to nitroprusside in the treatment of acute hypertension.[11] Both agents were found to have equivalent efficacy in reducing blood pressures; however, fenoldopam demonstrated improved creatinine clearance, urine flow rates, and sodium excretion.

In addition, DA_1 receptor agonists may have an added benefit in cardiac function. By providing a direct vasodilatory effect on myocardial vessels, the "cardiac steal" phenomenon is avoided and an

increase in myocardial blood flow is noted.[12] In conclusion, because of fenoldopam's rapid onset (usually within five minutes), short half-life (duration of action once discontinued is 30-60 minutes), and renal/cardiac effects, it is an excellent alternative for the treatment of hypertensive emergencies, especially in patients with impaired renal function. The dose of fenoldopam is based on body weight and titrated to desired effect with a starting point of 0.1 mg/kg/min.

Nicardipine

Nicardipine is a dihydropyridine-derivative calcium channel blocker that differs from nifedipine by the addition of a tertiary amine structure. Although very similar in structure, these changes make nicardipine 100 times more water-soluble than nifedipine and, therefore, it can be administered intravenously. The onset of action of nicardipine is 5-15 minutes, with a duration of action of roughly 4-6 hours; thus, this drug is an excellent candidate for the treatment of hypertensive emergencies.[8]

Multiple studies comparing the effects of nicardipine with nitroprusside on patients presenting with severe hypertension have demonstrated equivalent efficacy for lowering blood pressure. Intravenous nicardipine, however, has been shown to reduce both cardiac and cerebral ischemia.[8] Studies of both dogs and baboons also have demonstrated a significant cardioprotective effect of nicardipine on animal hearts with induced ischemia.[13] Nicardipine appears to be a good alternative for patients presenting with hypertensive emergency and evidence of acute myocardial ischemia.

Nicardipine's side effect profile is relatively safe, with headache, dizziness, flushing, reflex tachycardia, and excessive hypotension reported. The recommended dosage is 5 mg/hr, increasing the infusion by 2.5 mg/hr every five minutes (to a maximum of 15 mg/hr) until the required blood pressure reduction is achieved.

Summary

The management of hypertensive emergencies has advanced over the past couple of decades, due to a better understanding of its pathophysiology and an improved pharmacologic armamentarium. Given the significant side effect profile of sodium nitroprusside, physicians need to aggressively seek alternative treatment modalities that will be dictated by the organ system most in jeopardy. Doing so will perhaps contribute to decreasing mortality and morbidity in this disease. *(Dr. Botros is an Assistant Professor of Emergency Medicine at Temple University School of Medicine, and serves as the Assistant Research Director for Sponsored Projects within the Department of Emergency Medicine at Temple University Hospital.)* ❖

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To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

CME Questions

37. In patients with gunshot wounds to the abdomen:

a. CT scan routinely should precede operative management.
b. nonoperative management appears safe in patients who present with peritoneal signs, as long as broad-spectrum antibiotics are administered promptly.
c. selected patients can be managed nonoperatively, a practice which appears to decrease hospital length-of-stay and cost.
d. serial ultrasonography has replaced exploratory laparotomy as the initial assessment tool of choice in all anterior abdominal gunshot wounds.

38. Based upon the findings of the study on operative vs. non-operative management of abdominal gunshot wounds, which of the following patients could possibly be managed nonoperatively?

a. Anterior abdominal wound, hypotension, abdominal tenderness
b. Posterior abdominal wound, intoxicated, no prior abdominal surgery
c. Anterior abdominal wound, pain-free, old C5 spinal cord injury
d. Posterior wound, nonperitoneal abdominal examination, CT scan with non-threatening trajectory

39. From the data presented, first-trimester pregnant women who have trichomonas vaginalis infection probably should *not* be treated if they are:

a. nulliparous.
b. asymptomatic.
c. diabetic.
d. symptomatic.

40. The short-course, high-dose amoxicillin therapy for acute otitis media recently approved by the FDA is:

a. amoxicillin 40 mg/kg/day divided three times a day for 10 days.
b. amoxicillin 60 mg/kg/day divided twice a day for one week.
c. amoxicillin 90 mg/kg/day divided twice a day for five days.
d. amoxicillin 120 mg/kg/day divided three times a day for five days.

41. "Cardiac steal" is an undesirable side effect of which drug?

a. Nitroprusside
b. Nicardipine
c. Fenoldopam
d. Nitroglycerin

42. Fenoldopam seems to have protective effects on which one of the following organ systems?

a. Hepatic
b. Renal
c. Dermatologic
d. Pulmonary

How Many PACs?

By Ken Grauer, MD

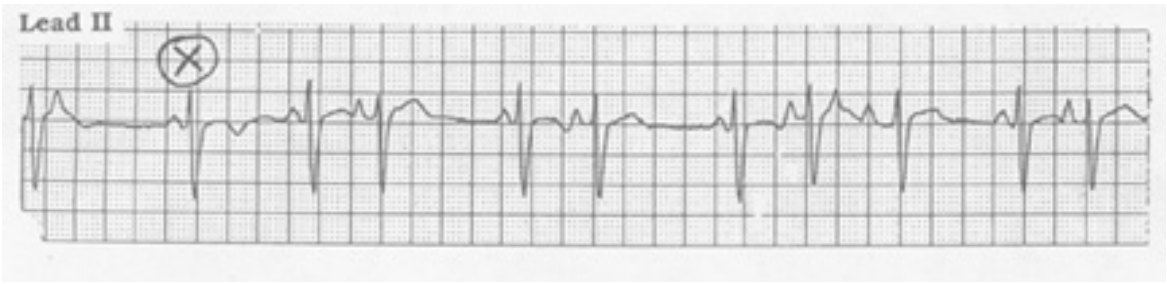

Figure. Rhythm strip showing PACs. How many PACs are there?

Clinical Scenario: The rhythm strip shows premature atrial contractions (PACs). How many PACs are seen on this tracing?

Interpretation: This is a tricky tracing to interpret. The starting point (and perhaps the most difficult part of the interpretation) is to determine what an unaffected sinus beat looks like. We propose that the beat marked X represents the only such unaffected beat on the tracing. Note that this beat manifests a rounded (coved) ST segment with a shallow, symmetrically inverted T wave. All other T waves on this tracing are distorted by premature P waves (PACs). Many of these PACs are blocked—some subtly (producing slight peaking in the T wave of the 4th, 6th, 9th, and 11th beats)—with other blocked PACs being much more obvious (note peaked PACs in the T waves of the very first beat on this tracing, as well as for the 8th beat). In all, we count a total of 11 PACs—but expect that others might not quite count the same. The answer is, therefore, that there are a lot of PACs on this tracing, with slight variation in their time of occurrence accounting for the continual change in ST-T wave morphology (due to superposition of these PACs on the normal T wave, which is negative in this lead). ❖

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	(4)	Other Classes Mailed Through the USPS	0	0
c. Total Paid and/or Requested Circulation *(Sum of 15b(1) and 15b(2)*			436	475
d. Free Distribution by Mail *(Samples, Complimentary and Other Free)*	(1)	Outside-County as Stated on Form 3541	0	0
	(2)	In-County as Stated on Form 3541	0	0
	(3)	Other Classes Mailed Through the USPS	0	0
e. Free Distribution Outside the Mail *(Carriers or Other Means)*			4	4
f. Total Free Distribution *(Sum of 15d and 15e)*			4	4
g. Total Distribution *(Sum of 15c and 15f)*			440	479
h. Copies Not Distributed			167	396
i. Total *(Sum of 15g, and h.*			607	875
Percent Paid and/or Requested Circulation *(15c divided by 15g times 100)*			99	99

16. Publication of Statement of Ownership
Publication required. Will be printed in the November issue of this publication. ☐ Publication not required.

17. Signature and Title of Editor, Publisher, Business Manager, or Owner: Publisher — Date: 9/27/01

I certify that all information furnished on this form is true and complete. I understand that anyone who furnishes false or misleading information on this form or who omits material or information requested on the form may be subject to criminal sanctions (including fines and imprisonment) and/or civil sanctions (including multiple damages and civil penalties).

Instructions to Publishers

1. Complete and file one copy of this form with your postmaster annually on or before October 1. Keep a copy of the completed form for your records.
2. In cases where the stockholder or security holder is a trustee, include in items 10 and 11 the name of the person or corporation for whom the trustee is acting. Also include the names and addresses of individuals who are stockholders who own or hold 1 percent or more of the total amount of bonds, mortgages, or other securities of the publishing corporation. In item 11, if none, check the box. Use blank sheets if more space is required.
3. Be sure to furnish all circulation information called for in item 15. Free circulation must be shown in items 15d, e, and f.
4. Item 15h., Copies not Distributed, must include (1) newsstand copies originally stated on Form 3541, and returned to the publisher, (2) estimated returns from news agents, and (3), copies for office use, leftovers, spoiled, and all other copies not distributed.
5. If the publication had Periodicals authorization as a general or requester publication, this Statement of Ownership, Management, and Circulation must be published; it must be printed in any issue in October or if the publication is not published during October, the first issue printed after October.
5. In item 16, indicate date of the issue in which this Statement of Ownership will be published.
6. Item 17 must be signed.

Failure to file or publish a statement of ownership may lead to suspension of second-class authorization.

PS Form **3526**, September 1999 *(Reverse)*

In Future Issues:

Chest Pain in Young Adults

Treatment of Biological Agent Exposure

AGENT	CLINICAL SIGNS AND SYMPTOMS	TREATMENT	OTHER	SECONDARY TRANSMISSION
Anthrax (spore)	Prophylaxis/treatment: Fever, malaise, non-productive cough, progressing to dypsnea, stridor, shock. Incubation 1-6 days.	Prophylaxis/treatment: ciprofloxin doxycycline, PCN licensed vaccine. IV therapy: ciprofloxin doxycycline, PCN licensed vaccine.	High mortality (>90%) even with treatment.	None except aerosolized body fluids.
Pneumonic Plague (bacteria)	High fever, chills, headache, hemophysis, toxemia, dyspnea, stridor, bleeding diathesis. Incubation 2-3 days.	Prophylaxis/treatment: vaccine, doxycycline, TMP/sulfamethoxazole. IV therapy: streptomycin (>1 yo), gentamicin, chloramphenicol.	Antibiotic treatment effective if begun early.	Strict isolation needed. Isolation mandatory for at least the first 48 hours of treatment.
Tularemia (bacteria)	Regional lymphademepathy, fever, chills, headache, malaise, cutaneous ulcers. Incubation 2-10 days.	Streptomycin, gentamicin. Adult prophylaxis: doxycycline.	Low mortality (about 5%).	Rare, body fluid precautions only.
Q Fever (bacteria)	Fever, cough, pleuritic chest pain. Incubation 10+ days.	Tetracycline, doxycycline.	Low mortality.	Does not require universal precautions.
Smallpox (virus)	Malaise, fever, rigors, vomiting, headache, backache; 2-3 days later lesions appear and quickly progress from macules to paupules to pustular vesicles. Incubation 16-17 days.	Supportive — vaccine available from CDC. Immune globulin may be available from CDC. No antiviral medication available.	Supposed to be extinct (doubtful).	Highly contagious.
Viral Equine Encephalitis	Supportive. No antiviral medication exists.	Ribaviron, supportive care.	Isolate patients in single room with an adjoining anteroom stocked with PPE. Negative air pressure if possible.	Body fluids. Otherwise infectious by vector (mosquitoes).
Viral Hemorrhagic Fevers	Fever, malaise, myalgias, headache, vomiting, diarrhea, easy bleeding, petechiae, shock.	Ribaviron, intensive care, convalescent plasma (Argentine HF), vaccine (yellow fever), blood replacement products for DIC.	Decontaminate with hypochloride or phenolic disinfectants.	Transmitted by bodily fluids. Strict barrier-nursing techniques. Limit patient transfers: may increase risk for secondary transmission.
Botulism (toxin)	Ptosis, weakness, dizziness, dry mouth, blurred vision, diplopia, descending paralysis. Incubation 24-36 hours.	Several antitoxins are available and effective if administered early. CDC vaccine good only for A and B.	Disinfect with hypochlorite and/or soap and water. Supportive long-term mechanical ventilation.	None.
Ricin (toxin)	Weakness, fever, cough, pulmonary edema, incubation 18-24 hours.	Supportive — oxygenation and hydration. No antitoxin or vaccine available.	Disinfect with hypochlorite and/or soap and water.	None. Derived from caster beans.
Staphylococcal Enterotoxin B (toxin)	Fever, headache, chills, myalgias, cough, nausea, vomiting, diarrhea, Incubation 3-12 hours.	Supportive — oxygenation and hydration. Ventilator support may be required.	Disinfect with hypochloride. Most victims recover.	Use PPE.

Source: Robert Suter, DO, MHA, FACEP, Questcare Emergency Services, Plano, TX.

In light of recent events, this special supplement is presented with our compliments as part of our continuing effort to provide you with the most up-to-date and useful medical information available.