Table 1 Results of a 10-laboratory collaborative study to reassess quality control limits for cefotaxime disk susceptibility tests

Control strain	No. zones evaluated[a]	Current quality control limits		Observed zone diameters	
		Range[b]	Midpoint	Median (mm)	% in range
Pseudomonas aeruginosa ATCC 27853	600	18–22	20	21	99.7%
Escherichia coli ATCC 25922	592	29–35	32	33	97.8%
Staphylococcus aureus ATCC 25923	580	25–31	28	28	98.6%
Haemophilus influenzae ATCC 49247	580	31–39	35	34	98.1%
Streptococcus pneumoniae ATCC 49619					
NCCLS quality control limits	598	30–35	32.5	35	60.2%
Proposed range	598	31–39	35	35	96.3%

[a] Of the 600 determinations, some tests were excluded because the control disk gave zones outside of the established control ranges: for different control strains, 3.3–0.3% of all cefotaxime zones were excluded. The control disk for the *Streptococcus pneumoniae* and *Haemophilus influenzae* strains was cefixime, whereas cefaclor was the control disk for the *Staphylococcus aureus* and *Escherichia coli* strains. A pseudomonas-active control disk could not be included because of space limitations. [b] Range of acceptable zone diameters (mm) listed in the current NCCLS document, M100-S9 [2]. For *Streptococcus pneumoniae* ATCC 49619, an alternative control range is also listed. The midpoint between the upper and lower limits is an idealized target value.

four of the five control strains were reconfirmed, since 98.1–99.7% of the 600 zones fell within the established ranges and the modal values were within 1 mm of the midpoint between the two extremes. This included a narrow 5-mm range for *P. aeruginosa* ATCC 27853 and a broader 9-mm range for *H. influenzae* ATCC 49247. However, when *S. pneumoniae* ATCC 49619 was tested on Mueller–Hinton blood agar, only 60% of the zones were within the narrow 6-mm range of 30–35 mm, and the modal value was at the outer limit of 35 mm. The current zone size limits are clearly inappropriate, and we propose a broader range of 31–39 mm for the *S. pneumoniae* control strain. For that strain, other extended-spectrum cephalosporins have 7- or 8-mm ranges [2], and this is compatible with the 9-mm range that we are proposing for cefotaxime disk tests. Our proposed change in zone size limits has recently been approved by the NCCLS subcommittee on antimicrobial susceptibility tests, and the new limits will appear in future NCCLS publications. This report describes the data that supported that change in QC limits.

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Ascites and pleural effusion accompanying hepatitis A infection in a child

Up to 1992, seven children were reported with acute hepatitis A associated with ascites [1–3]. Pleural effusion also represents a rare complication of acute type A viral hepatitis [4]. I report a recent observation on a rare presentation of both ascites and pleural effusion accompanying hepatitis A infection in a child.

A 4-year-old boy was hospitalized because of abdominal distension and jaundice of the skin and sclerae. Four days earlier,

he complained of headaches, anorexia, abdominal pain and vomiting, and he developed a mild-to-moderate fever 2 days later.

On initial physical examination, his temperature was 38.5 °C, his heart rate was 98 beats/min, and mild jaundice of the skin and sclerae was present. The abdomen was distended, and fluid was detected by abdominal percussion. The liver was palpable 4 cm below the costal margin, but the spleen was not palpable. There were no other abnormal findings in the physical examination.

Laboratory values were as follows: total bilirubin 6 mg/dL, direct bilirubin 2.4 mg/dL, aspartate aminotransferase 3660 U/L, alanine aminotransferase 2855 U/L, alkaline phosphatase 1306 U/L, lactate dehydrogenase 1010 U/L, urea nitrogen 23 mg/dL, creatinine 0.6 mg/dL, total protein 6.0 g/dL, albumin 3.6 g/dL, sodium 134 mmol/L, potassium 4.1 mmol/L, serum amylase 22 U/L, α_1-antitrypsin 3.8 g/L (normal range 2.0–4.0 g/L), and ceruloplasmin 300 mg/L (normal range 150–550 mg/L). The results of urine analysis showed a +3 value for bilirubin, increased levels of urobilinogen but no protein. Sediment was normal. Both anti-HAVIgM and anti-HAVIgG antibodies were detected. Hepatitis B surface antigen and antibodies to hepatitis B, C and E were absent. Serologic analysis for cytomegalovirus and Epstein–Barr virus revealed no evidence of recent infection. Bilateral moderate pleural effusions were shown on chest X-ray on the sixth day of hospitalization but were not detected on initial chest radiogram. Abdominal ultrasonography showed ascites. An ultrasound study 15 days later showed complete resolution of both ascites and the pleural effusion. Levels of liver transaminases became normal 1 month later and the child recovered completely.

Ascites in liver diseases may occur as a result of venous and lymphatic obstruction or decreases in the osmotic pressure of plasma colloid, such as in hypoalbuminemia. A transient increase in portal venous or lymphatic pressure due to the compression of hepatic sinusoids may explain the occurrence of ascites in this case. Pleural effusion may be secondary to ascites due to fluid transport through the diaphragmatic lymphatics or direct passage through a diaphragmatic defect. Our patient developed bilateral pleural effusion 6 days after the diagnosis of ascites associated with type A viral hepatitis.

In conclusion, ascites and pleural effusion may exceptionally accompany hepatitis A infection in children, and presumably the same may occur in adults. Its appearance, however, does not indicate an unfavorable outcome.

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Disseminated *Nocardia asteroides* complex infection in an immunocompromised child

Nocardiosis is a rather frequent infectious disease that usually involves the respiratory tract [1–5]. Predisposing factors include malignancies, AIDS, organ transplantation, tuberculosis, drugs or alcohol abuse, lupus erythematosus, diabetes and chronic lung disease [2,4]. Very few cases have been reported in Greece, therefore we report a case of pulmonary nocardiosis in an 8-year-old immunocompromised female.

The girl was admitted to our hospital because of fever and cough. She had been suffering from autoimmune hemolytic anemia and thrombopenia (Evans's syndrome) from the age of 1 year, receiving corticosteroids and cyclosporin periodically. A *Mycobacterium avium* complex (MAC) disseminated infection had taken place a year ago, which was treated with a combination of ethambutol, rifampicin, cycloserine and clarithromycin.

On admission, the laboratory studies showed WBC count of 27800/μL, with 87% neutrophils, haemoglobin 17.6 g/dL, erythrocytes sedimentation rate (ESR) 15 mm/h, CRP 55 mg/L, AST 62 U/L and ALT 64 U/L, urine protein 300 mg/dL with a few granular and rare waxy casts in the urinalysis. Blood and urine cultures were sterile and exclusive growth of *Candida albicans* was found in fecal cultures. The main roentgenographic findings included a centrally placed circumscribed shadow in the left middle lobe with mediastinal lymph node enlargement to the same side. *Candida albicans* was isolated in urine culture 2 weeks after the admission. Some subcutaneous scalp abscesses were noted 1 month after the admission and the ESR had increased to 110 mm/h.

Nocardia asteroides complex (NAC) was then isolated from blood, sputum and the exudate of the subcutaneous abscesses. The initial Gram stain preparations revealed Gram-positive, beaded, partially acid-fast, branched filaments. An empirical course of treatment was given, which consisted of amikacin, trimethoprim-sulfamethoxazole and ceftriaxone. In the following days a final identification of NAC was made, based on colony morphology and biochemical characteristics [4]. The susceptibility testing was performed by a modified disk diffusion