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## Dr. Gerard “Jerry” Boe – 1936-2016



We are saddened to report that Dr. Gerard “Jerry” Boe of Beaufort, SC, died on October 22, 2016, after a long illness. Dr. Boe served as Executive Director of AMT from 1989 to 2000, and was the Editor of AMT’s *Journal of Continuing Education Topics & Issues*.

After his retirement from AMT in 2000, he continued to be active in AMT by remaining as Executive Director of AMTIE, in addition to continuing as the former Liaison to the Caribbean Association of Medical Technologists (CASMET) and AMT’s representative to the Coalition for Professional Certification.

He was instrumental in the expansion of AMT during his tenure, both in membership and programs. He grew membership over a period of a decade to an all-time high for AMT at the time. He raised certification standards, revising the entry-level MT qualification requirement to a Bachelor’s degree.

He was also instrumental in the implementation of the former COLT and CLC certification designations; he initiated the establishment of the AHI membership designation; he initiated the establishment of the STEP program over the Internet; he promoted the transition to computer-administered testing; he developed significant rapport and linkages with the Caribbean Association (CASMET); and he conducted four Annual Forums on Medical Laboratory Personnel Standards.

Dr. Boe initiated operational changes in the AMT office which resulted in financial gain for AMT (AMT remained in a positive financial position each year of his tenure). Some of the changes he initiated were revamping the accounting and payroll system from manual to computerized; writing operating, salary and administration policies; satisfying the mortgage on the AMT building; and developing a five-year strategic plan. In 1999, he also founded AMT’s *Journal of Continuing Education Topics & Issues* as an independent publication; before this time, it was just a supplement to AMT’s other magazine *AMT Events*.

He earned a BS degree in Biology from West Virginia Wesleyan College; MS in Clinical Pathology from Ohio State University; and PhD in Allied Health Teacher Education and Administrative Leadership from Texas A&M University.

He taught science and medical technology at several colleges and universities. He published articles widely and presented papers and workshops at many professional meetings. Certified as a medical technologist by AMT in 1983, he served on the Georgia state society Board of Directors.

He spent 21 years in military service. Dr. Boe graduated from the US Marine 26th OCC at Quantico, VA, and served his initial tour of duty as an Air Intelligence Officer at MCAS, Beaufort, SC. He transferred into the US Army Medical Service Corps where he held both staff and supervisory positions including Laboratory Officer, 3rd Field Hospital Wurzburg, Germany; OIC, Vietnam (USARV) Central Blood Bank; Chief, Special Subjects Branch; Chief, Hematology Branch at the Academy of Health Sciences; and Administrative Chief, Dept. of Pathology, Dwight David Eisenhower Army Medical Center.

He was a founding member of the Society of Air Force Medical Laboratory Scientists (SAFMLS), and the first Army Officer to be admitted. He was National President of the International Society for Clinical Laboratory Technology; and Chairman, Board of Regents, Institute of Certified Professional Managers.

Among his many military honors and awards were: For federal service – the Bronze Star; Meritorious Service Medal; Army Commendation Medal with Oak Leaf; National Defense Medal; Vietnam Service Medal; and Vietnam Gallantry Medal. For his service with the South Carolina State Guard, he received the Service Ribbon; Military Readiness Ribbon; Emergency Service Training Ribbon; and the Cold War Ribbon.

He is survived by his wife Charlotte, son Steven, daughter Christine, step-daughter Kimberly Kinane, five grandchildren, and sister Nora Lankford. Graveside services with full military honors were held.

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*On the cover: 3D representation of a Zika virus*

Zika virus was first discovered in 1947 and is named after the Zika Forest in Uganda. In 1952, the first human cases of Zika were detected and since then, outbreaks of Zika have been reported in tropical Africa, Southeast Asia, and the Pacific Islands. Zika outbreaks have probably occurred in many locations. Before 2007, at least 14 cases of Zika had been documented, although other cases were likely to have occurred and were not reported. Because the symptoms of Zika are similar to those of many other diseases, many cases may not have been recognized. (*Zika Virus Overview, Centers for Disease Control*)

*Image Credit:* Manuel Almagro Rivas, <https://commons.wikimedia.org/wiki/File:Zika-virus-3D.png>

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# Zika Virus, Past and Present

*By Lisa Maness and Jeffery Meixner*

## Introduction

Zika Virus (ZIKV) had been virtually unheard of in the U.S. before resonating throughout the press in 2016. At this time, one heard that it correlated with mosquitoes in Brazil causing birth defects. People wanted proof that ZIKV causes these issues, and scientists began to work toward diagnosing and treating the disease.

ZIKV has an interesting history, beginning in the Zika Forest in Uganda and spreading gradually toward Asia before its appearance in South America.<sup>1-3</sup> Although symptoms of Zika cases varied, none were previously documented to have caused birth defects or deaths before 2016.<sup>4,5</sup> It was simply reported as being similar to other arboviruses, such as Dengue Virus (DENV) and West Nile Virus.

Different species of mosquitoes from the *Aedes* genus carried and transmitted the virus with shifts in species occurring based on the predominant *Aedes* mosquito in the region in which the virus is spreading.<sup>1,6</sup> ZIKV was first isolated from *A. africanus*, and thereafter was found in other species. This same genus is also responsible for transmitting other arboviruses that many people are unfamiliar with.

There are an extensive number of arboviruses throughout the world.<sup>7</sup> While most are carried by mosquitoes, ticks and flies are other vectors of arbovirus spread. Examples of other arboviruses include DENV, Chikungunya, Rift Valley Fever, LaCrosse, Eastern Equine Encephalitis, Yellow Fever Virus (YFV), and Crimean-Congo Hemorrhagic Fever. ZIKV itself is a single stranded RNA virus that belongs to the *Flaviviridae* family,

along with YFV, West Nile Virus, Japanese Encephalitis Virus, and DENV.

## Zika's path

The name, Zika, came from the forest where the virus was discovered in Uganda in 1947 (Table 1).<sup>1</sup> It was first isolated from an experimental Rhesus monkey with a fever and no other abnormalities after a month of observation. A serum sample taken from this monkey was injected intracerebrally into mice and all of them became sick on the 10th day. In 1948, researchers were attempting to isolate the YFV from mosquitoes in the Zika Forest. Instead of YFV, ZIKV was isolated from *A. africanus* mosquito suspensions, which were then injected into a Rhesus monkey. Blood from this monkey injected into mice made some of them sick, depending on the day the blood was drawn from the monkey.

In 1954, three patients were reportedly infected with ZIKV in Nigeria, which is West of Uganda.<sup>4</sup> The three cases had variable symptoms, among which were cough, joint pain, jaundice, fever, loose bowels, and headache. Each of these patients exhibited neutralizing antibodies to ZIKV, and the virion itself was said to have been isolated from one. However, this claim was doubted by Simpson (1964), who reported that the virus responsible for these cases was actually Spondweni Virus.<sup>8</sup> Simpson went on to claim that he had experienced the first human case of ZIKV, with symptoms of headache, rash, and fever and a coinciding rise in ZIKV antibodies after onset of illness. Interestingly, around the

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**1 Clock Hour**

# Case Thirty-One: Need For Speed: When Turnaround Time Matters

*By Scott Bielewicz, Heidi Andersen,  
Rebecca Brady, and Joel Mortensen*

**EDITOR'S NOTE:** BEFORE reading the Case Follow-up and Discussion below, study the Case Description on page 2 of this issue, and formulate your own answers to the questions posed.

The bioMérieux MALDI-ToF MS Plus (bioMérieux, Laurent, Kansas) was used for identification of the isolate. The In-Vitro Diagnostic (IVD) database gave an identification of *Nocardia otitidiscaviarum* with a confidence of 96%. The same identification and confidence result was obtained using the SARAMIS research database. The isolate was inoculated onto a trypticase soy agar slant and sent to The University of Texas Center at Tyler Mycobacteria/Nocardia Laboratory for identification by 16S ribosomal RNA gene sequencing and antimicrobial susceptibility testing.

Because the Gram-stain of her sputum on HD 8 was consistent with a possible *Nocardia* infection, coverage with clindamycin was discontinued, high dose trimethoprim-sulfamethoxazole (5 mg/kg IV every 6 hours) was added to an increased dose of ceftriaxone (50 mg/kg IV every 12 hours) for appropriate coverage. Due to the patient's headaches there was concern for possible central nervous system involvement. On HD

11, a bronchoalveolar lavage (BAL) specimen demonstrated many beaded Gram-positive rods which were similar to those observed from her previous sputum specimen. She was continued on high dose trimethoprim-sulfamethoxazole but transitioned from meningitic dosing of ceftriaxone to IV meropenem (40 mg/kg every 8 hours) for more targeted therapy directed at *Nocardia spp.*, which are typically susceptible to carbapenems.

On HD 13, the patient had a magnetic resonance imaging (MRI) of the brain with and without contrast, which showed an ill-defined T2/FLAIR hyperintense mass (a bright spot on the MRI image) with adjacent edema in the right high peripheral parietal region with lace-like nodular enhancement. In addition, multiple T2/FLAIR hyperintense signal foci were noted in the bilateral white matter (Figure 2b). These observed abnormalities were consistent with a possibly bacterial infection located within the mid and posterior regions of the brain. On HD 19, the reference laboratory confirmed an identification of *Nocardia otitidiscaviarum* by 16S rRNA gene sequencing and released susceptibility results. The susceptibility report allowed her antimicrobial therapy to be transitioned to more definitive treatment consisting of high dose trimethoprim-sulfamethoxazole and IV linezolid

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# Hematology Case Study: A Microcytic, Hypochromic Anemia

By *Georgia A. McCauley*

A 22 year old college student was experiencing extreme fatigue. Since she had a history of anemia and intermittent blood streaked stool, she decided to visit her primary care physician. The physician ordered a complete blood count (CBC) with differential. The results can be seen below. The CBC report was flagged by the automated instrument for a peripheral blood smear (PBS) review. The image below is from the Giemsa-Wright stained PBS (x1000).

### Guided Questions & Discussion:

1. What are the implications of the complete blood count parameters and peripheral blood smear (PBS) image seen in this case study?

Parameter	Patient Results	Reference Intervals (8)
WBC	6.5	3.6 – 10.6 x 10 <sup>3</sup> /uL
RBC	3.48 L	3.80 – 5.20 x 10 <sup>6</sup> /uL
HGB	5.3 L	12.0 – 15.0 g/dL
HCT	20.1 L	35– 49 %
MCV	57.9 L	80 – 100 fL
MCH	15.2 L	26 – 31 pg
MCHC	26.2 L	32 – 36 %
RDW	21.1 H	11.5 – 14.5 %
PLTC	687 H	150 – 450 x 10 <sup>3</sup> /uL
MPV	8.9	7.0 – 12.0 fL
<b>WBC Differential:</b>		
Neutrophils	66.3	50 – 70 %
Lymphocytes	28.3	18 – 42 %
Monocytes	4.2	2 – 11 %
Eosinophils	1.0	1 – 3 %
Basophils	0.2	0 – 2 %
NEUT #	4.3	1.7 – 7.5 x 10 <sup>3</sup> /uL
LYMPH #	1.8	1.0 – 3.2 x 10 <sup>3</sup> /uL
MONO #	0.3	0.1 – 1.3 x 10 <sup>3</sup> /uL
EOS #	0.1	0 – 0.3 x 10 <sup>3</sup> /uL
BASO #	0.0	0 – 0.2 x 10 <sup>3</sup> /uL

The white blood cell (WBC) count is within the reference range or interval for the age and gender of the patient.<sup>8</sup> The red blood cell count (RBC), hemoglobin (HGB), and hematocrit (HCT) are decreased from the reference intervals indicating anemia, resulting in decreased oxygen carrying capacity of the red blood cells. The HGB is 5.3 g/dL which is significantly lower than the reference, therefore the anemia is severe.<sup>5,7,8</sup> Applying the rule of three ( $HGB \times 3 = HCT \pm 3$ ) to the hemoglobin and hematocrit values indicates that the two parameters do not correlate. However, the rule of three, a fast mathematical accuracy check that could indicate instrument error or abnormal red blood cells, can only be applied to normocytic, normochromic erythrocytes.<sup>5,8</sup> In this case, the RBC indices, mean cell volume (MCV), mean cell hemoglobin (MCH) and the mean cell hemoglobin concentration (MCHC) are all decreased. This represents a microcytic and hypochromic red cell population, according to the morphologic classification of anemia.<sup>5,7,8</sup> An elevated red cell distribution width (RDW) means that there is anisocytosis, or varying cell diameters in the RBC population. The platelet count (PLTC) is also significantly increased. Primary or second-

Figure 1



(Image courtesy of Stephanie Nesmith, BS, MLS(ASCP) CM, Winston-Salem State University)

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