

Archived version from NCDOCKS Institutional Repository <http://libres.uncg.edu/ir/asu/>



LITTLE ALBERT: A Neurologically Impaired Child

Authors: Alan J. Fridlund, William D. Goldie, Gary Irons, **Hall P. Beck**

Abstract

Evidence collected by Beck, Levinson, and Irons (2009) indicates that Albert B., the “lost” infant subject of John B. Watson and Rosalie Rayner’s (1920) famous conditioning study, was Douglas Merritte (1919 –1925). Following the finding that Merritte died early with hydrocephalus, questions arose as to whether Douglas’s condition was congenital, rather than acquired in 1922, as cited on his death certificate. This etiology would imply that “Little Albert” was not the “healthy” and “normal” infant described by Watson and numerous secondary sources. Detailed analyses of Watson’s (1923) film footage of Albert suggested substantial behavioral and neurological deficits. The anomalies we observed on film of Albert B. are insufficiently explained by his hospital upbringing but are consistent with findings from newly discovered medical records of Douglas Merritte. These documents revealed that the infant suffered from congenital obstructive hydrocephalus, iatrogenic streptococcal meningitis/ventriculitis, and retinal and optic nerve atrophy. The medical history also indicates that Albert’s sessions with Watson occurred during periods when Douglas’s clinical course was relatively stable. Further inquiries found ample sources of information available to Watson that would have made him aware of Douglas/Albert’s medical condition at the times he tested the baby. Experimental ethics, Watson’s legacy, and the Albert study are discussed in light of these new findings.

Alan J. Fridlund, William D. Goldie, Gary Irons, Hall P. Beck(2012) LITTLE ALBERT:
A Neurologically Impaired Child. *History of Psychology* Vol. 15, No. 4, 302–327 (DOI:
10.1037/a0026720) (ISSN: 1093-4510)

LITTLE ALBERT: A Neurologically Impaired Child

Alan J. Fridlund

University of California, Santa Barbara

Hall P. Beck

Appalachian State University

William D. Goldie

University of California, Los Angeles and
University of Southern California

Gary Irons

Finksburg, Maryland

Evidence collected by Beck, Levinson, and Irons (2009) indicates that Albert B., the “lost” infant subject of John B. Watson and Rosalie Rayner’s (1920) famous conditioning study, was Douglas Merritte (1919–1925). Following the finding that Merritte died early with hydrocephalus, questions arose as to whether Douglas’s condition was congenital, rather than acquired in 1922, as cited on his death certificate. This etiology would imply that “Little Albert” was not the “healthy” and “normal” infant described by Watson and numerous secondary sources. Detailed analyses of Watson’s (1923) film footage of Albert suggested substantial behavioral and neurological deficits. The anomalies we observed on film of Albert B. are insufficiently explained by his hospital upbringing but are consistent with findings from newly discovered medical records of Douglas Merritte. These documents revealed that the infant suffered from congenital obstructive hydrocephalus, iatrogenic streptococcal meningitis/ventriculitis, and retinal and optic nerve atrophy. The medical history also indicates that Albert’s sessions with Watson occurred during periods when Douglas’s clinical course was relatively stable. Further inquiries found ample sources of information available to Watson that would have made him aware of Douglas/Albert’s medical condition at the times he tested the baby. Experimental ethics, Watson’s legacy, and the Albert study are discussed in light of these new findings.

Keywords: Watson, Little Albert, ethics, history of psychology, behavior therapy

This article is an unplanned sequel to a report by Beck, Levinson, and Irons (2009), which detailed a 7-year quest to find the historical “Little Albert.” As almost every psychology student learns, Watson and Rayner (1920) at-

tempted to condition an 11 month-old baby they called “Albert B.” to fear a white laboratory rat. They contended that the child’s fears later transferred to other furry objects, such as a rabbit, a dog, and a Santa Claus mask.

The continued interest in Little Albert is due, in part, to questions that Watson and Rayner (1920) left unanswered about him. Albert departed from his home at The Johns Hopkins University campus (hereafter referred to as Johns Hopkins) without deconditioning or long-term follow-up. His departure created one of the greatest mysteries in our discipline, generating lingering questions that have intrigued generations of psychologists. Did he retain his fear of furry objects? Did he develop other phobias as an adult? What type of person did he become? In the absence of reliable information, Albert became the source of speculations, myths, and misconceptions (see Harris, 1979).

The final paper (Beck et al., 2009) presented some new facts about Albert, which were supplemented with material from Watson's scientific productions (books, journal articles, and personal correspondence). The most important information reported by Beck et al. (2009) was that (a) Albert's mother was one of no more than four wet nurses employed at the Johns Hopkins Harriet Lane Home for Invalid Children (HLH), (b) he lived almost his entire first year at the HLH, and (c) he was born at Johns Hopkins between March 2 and March 16, 1919.

Examinations of census, birth, death and other public records led to a single child, Douglas Merritte, who had these and a number of other attributes. Contact with Douglas's family generated additional information. The most significant was a photographic portrait of Douglas as a baby. Biometric analyses comparing stills of Albert and Douglas's portraits revealed substantial facial similarities (Beck et al., 2009). These and other findings led Beck et al. (2009) to conclude the following:

It is possible, but improbable, that these commonalities are happenstance. Although some of these attributes apply to more than one person, the likelihood that the entire set applies to anyone other than Albert is very small. The available evidence strongly supports the hypothesis that Douglas Merritte is Little Albert. (Beck et al., 2009, p. 612)

When Beck et al. (2009) submitted their manuscript, they assumed that the major sources of information about Albert had been exhausted and that their narrative reasonably accounted for the available data. This conclusion proved premature. Following the publication of Beck et al. (2009), two new colleagues, Alan J. Fridlund and William D.

Goldie, reexamined final sequences of Albert (Watson, 1923). Their analyses yielded insights into the child's psychological and neurological condition. Then, Gary Irons, Douglas's closest living relative, learned new details about his uncle's medical history. These discoveries led us to a troubling but more complete view of the Little Albert study and the infant at its center.

Finding Little Albert: Reopening Our Inquiry (Beck)

The hunt for psychology's lost boy brought me (Beck, 2011), Gary Irons, and Gary's wife Helen to a modest tombstone in a rural Maryland graveyard. It read, "Douglas Merritte, Son of Arvilla Merritte, March 9, 1919 to May 10, 1925." The boy's death did not attract much attention, not even a mention in the local newspaper. Douglas left behind few clues by which we might know him—only a portrait, a bootie, a glove, and a card printed for his funeral.

Dr. Alan Crunk, the local physician who last attended Douglas, cited "hydrocephalus" and "convulsions" as the causes of death (Department of Health, Bureau of Vital Statistics, 1925). Crunk reported that Douglas contacted hydrocephalus in 1922, two years after the Little Albert study was conducted. Watson and Rayner (1920) attested to Albert's excellent physical development and emotional stability during his stay at the HLH. The data points seemed to align. It seemed reasonable to assume that Douglas became ill after leaving Johns Hopkins.

Crunk's contention that the onset of Douglas's disease was in 1922 was buttressed by an additional discovery. After leaving Johns Hopkins, Arvilla and Douglas moved in with the Brashears family near Mt. Airy, Maryland. Flora, the lady of the house, suffered from meningitis, a contagious disease that can cause hydrocephalus (Sarco, Vanderbilt, & Riviello, 2008).

Although we found no document contradicting Crunk's notations, I was not fully convinced that Douglas was healthy when he was tested by Watson and Rayner. The source of my uncertainty was Watson's (1923) motion picture, *The Experimental Investigation of Babies*. In the film, Albert's unresponsive nature reminded me

of many severely mentally challenged children I worked with earlier in my career.

My doubts were shared by members of the Irons family. Gary Irons learned from his mother that Douglas “always had problems.” Several months after publication of the Beck et al. (2009) article, Gary’s sister, a registered nurse, asked if I was sure that Douglas’s hydrocephalus was acquired (G. Merek, personal communication, December 16, 2009). I replied that we depended on the best available information when we wrote the manuscript. I did not believe we could give our unconfirmed suspicions precedence over the notes of a physician who saw Douglas the day before he died.

Still, somewhat defensively, I acknowledged that she and Gary might be right. Douglas’s hydrocephalus might have been congenital (H. P. Beck, personal communication, December 28, 2009). If Douglas had congenital hydrocephalus, a rare disorder with an incidence of one or two cases per 1,000 births (Avellino, 2005; Sarco et al., 2008), it would have implied that he had suffered for most of his short life. It also led to the uncomfortable possibility that Watson either knowingly or unknowingly induced fear in a neurologically damaged child. Six months later I received an e-mail from Alan Fridlund sharing his identical concern (A. J. Fridlund, personal communication, June 16, 2010). Fridlund’s e-mail initiated the collaboration that resolved the mystery of Douglas’s hydrocephalus and compelled a reevaluation of both the Little Albert study and Watson’s legacy to psychology.

This article details the results of that collaboration. The first part of this report describes how three sources of information—the appearance of Albert on Watson’s (1923) film, the Irons family history, and Douglas Merritte’s medical records—were brought to bear on Watson and Rayner’s (1920) claim that “Albert’s life was normal: he was healthy from birth and one of the best developed youngsters ever brought to the hospital” (p. 1).

Little Albert: A “Normal, Healthy” Infant? (Fridlund)

I learned of the Beck et al. (2009) article in May 2010, and I was moved by the immense array of evidence the authors brought forth in their search for Little Albert. I kept ruminating

about one section, however. This was the report that Douglas Merritte died in May 1925, after contracting hydrocephalus in 1922. Beck et al.’s (2009) speculation that Douglas developed hydrocephalus after being exposed to meningitis at the Brashears’s home was conceivable but implausible. It required Douglas to have been infected with a strain of meningitis sufficiently virulent to cause hydrocephalus, yet mild enough for him to survive for 3 years in a time before antibiotics or antivirals.

A chain of questions arose: What explained Douglas’s hydrocephalus if not the meningitis? Can we assume that Douglas acquired the condition in 1922 because that date was cited on his death certificate? If not, could he have been hydrocephalic when Watson and Rayner (1920) tested him? At first, the notion seemed preposterous. After all, those investigators had made a point of testifying to Albert’s excellent health and disposition.

The “normal, healthy” theme is recurrent among secondary sources: For example, an introductory text read, “‘Little Albert’ was a normal, healthy, well-developed infant” (Kassin, 2003, p. 177); a developmental text stated, “The subject of the experiment was Albert, a healthy, normal infant of nine months” (Vander Zanden, 1981, p. 66); a learning and behavior text claimed, “Little Albert was a normal, healthy infant” (Pierce & Cheney, 2004, p. 14); and a history of psychology text restated, “Albert was a normal, healthy child” (Feist & Feist, 2005, p. 441). Most recently, Harris (2011) referred to Albert as “the healthy baby that Watson filmed and showed to the world” (p. 3).

But what if Albert was not the healthy and well-developed boy described by Watson and Rayner (1920)? Then how should we interpret his emotional stability and his response to the experimental stimuli? According to Watson and Rayner (1920), Albert was “on the whole stolid and unemotional” (p. 1). Watson (1930), ever the descrier of disorderly sentimentality, praised Albert’s temperament (and, slyly, his own experimental prowess), declaring that Albert was a “wonderfully good baby. In all the months we worked with him we never saw him cry until after our experiments were made!” (p. 159).

“Emotional stability,” especially in an infant, can mean many things. Temperaments vary from infant to infant (e.g., Kagan, 2001), but given that Albert B. was most likely Douglas

Merritte, what are we to make of a baby who was stolid and unemotional, rarely cried, never displayed rage or fear, and died with hydrocephalus at age 6? Neurological normalcy is only one possibility. The issue of Albert's health and normalcy is crucial, because it bears on whether the child was a suitable participant for Watson and Rayner's (1920) procedures as well as the strength of their study's conclusions.

Film Analysis of Albert B. (Fridlund)

Having graduated from a behavioral clinical training program approximately 30 years ago, I had seen stills of Little Albert. Now, hoping to learn more, I downloaded JPEGs from the Internet. I then went to Google Video and found segments from *The Experimental Investigation of Babies* (Watson, 1923). Watching Albert in motion for the first time and, with benefit of age, clinical experience, and my own intervening fatherhood, I was struck by his general unresponsiveness, a passivity so apparent that I could not believe this was the same "normal, healthy" Albert routinely touted in textbooks.

In mid-June 2010, I e-mailed Beck (A. J. Fridlund, personal communication, June 16, 2010) and asked how confident he was that Douglas was not hydrocephalic before 1922. To my surprise, Beck told me that he and Douglas's family had doubts about the late onset of the disorder. Beck further confided the suspicions he harbored about the filmed Albert's neurological intactness. He welcomed my involvement in pursuing the Albert story to the next step, and we began our collaboration.

I suggested that the behavioral clues provided by Albert on Watson's (1923) film might be sufficiently pathognomonic to suggest a psychopathological or neurological diagnosis. Additionally, because some, but not all, cases of congenital or early developing hydrocephalus are marked by bulging fontanelles (soft spots between immature, unfused cranial bones) or age-discordant head circumference (Amiel-Tison & Gosselin, 2001), I also proposed that these might be observable photographically. Finally, I recommended combining my behavioral and photogrammetric analyses with an independent assessment of the film by a pediatric neurologist.¹ Beck agreed and sent me his own, clearer direct-to-DVD transfer of Watson's

(1923) film, made from Ben Harris's 16-mm print.

The video runs 24,000 frames (at 30 frames/s, or fps, for a total of 13 min, 20 s). Albert appears in four segments, for a total of 4 min. By comparing the film to Watson and Rayner's (1920) procedural descriptions, Beck determined that the first two segments were recorded when Albert was 8 months, 26 days old, and the final two segments were filmed when Albert was 11 months of age.²

Behavioral Analysis (Fridlund)

In the film segments, Albert is confronted with, and reacts to, various stimuli (blocks, animals, etc.). Watson is at the infant's right, presenting the stimuli; Rayner is behind Albert or at his left, often with her hands at the baby's back, holding him upright and/or orienting him to face the camera. The investigators do not tell us if the camera is attended or unattended. Nor were the times provided during which the sessions were run (e.g., before or after Albert's feedings or naptimes). Watson and Rayner (1920) do report that, at one point, four unidentified people were in the room watching Albert play with blocks. The film titles declare that Albert had no experience with blocks, crayons, fire, or animals, but there is no mention of what experiences he did have. These omissions made it more difficult to interpret Albert's reactions to the various events on the film.

Some potentially informative tests cannot be conducted because Albert is not available "in the flesh." His hearing, eyetracking, cranial nerves, Babinski reflexes, deep tendon reflexes, and muscle tone cannot be assessed directly (Amiel-Tison & Gosselin, 2001; Cohen & Duffler, 2003). There is no way to tickle him or blow on his belly to see if he smiles and giggles. It is difficult to ascertain whether he makes eye contact with people, babbles, chirps, gurgles, or yells "ma-ma." Neither can controls be run that are matched for age and psychosocial histories.

The evaluation of Albert was confined to his filmed behaviors, comparing them with age norms, noting obvious abnormalities, and then asking whether his overall presentation concerns or reassures. When making these determinations, it was necessary to consider that Albert was reared institutionally, not in a typical home environment. With these caveats in mind, I

viewed the Albert footage repeatedly, first at regular speed, then in slow motion, and, at times, frame-by-frame. While doing so, I tabulated the areas in which Albert's presentation was anomalous. The issues that emerged are described in the next several sections.

General Unresponsiveness; Lack of Aversive Recoil

The infant described by Watson and Rayner (1920) as "stolid and unemotional" (p. 1) looks alarmingly unresponsive. He appears dazed and slowed; his reaction times are "off." Stimuli moving rapidly around his face (e.g., the dog's head, the monkey cavorting at the end of a leash) produce weak, delayed tracking. Other stimuli thrust rapidly within inches of Albert's face (e.g., newspaper on fi Watson wearing a Santa Claus mask) elicit little startle responding such as head retraction or wincing. During nearly all of the aversive stimulus presentations—even the later ones, conducted when he was 11 months old—Albert's arms fl at his sides, being used neither for protection nor escape. He employs his arms only when he lurches forward onto them in attempt to crawl away. By 11 months of age, most infants are able to use their arms, legs, feet, tears, and words to manage aversive stimulation (e.g., Thompson, Easterbrooks, & Padilla-Walker, 2003). This hyporeactivity may indicate low arousal generally and/or compromised visual perception.

Amimia, Particularly the Lack of Social Smiling

With the exception of a "cry face," which we discuss as part of his language development, Albert is facially and gesturally impassive throughout the film. His eyebrows do not knit when he focuses on objects, nor do they "flash" when an object looms. His orofacial muscles are mask-like for most of the film. Not once does Albert smile during the 4 min of footage, despite the presence of at least two adults who are animated and smiling at times during much of the filming. (Curiously, Watson and Rayner, 1920, reported that Albert smiled twice while playing with blocks at 11 months, 15 days old, but they captured neither smile on film.)

Normally, by 9 months of age, babies show a wide range of facial expressions; these expres-

sions declare behavioral intentions and recruit the attentions of peers and adults (Fridlund, 1994). Social smiling, in particular, is expected to appear by 2 months of age (e.g., Sroufe & Waters, 1976), and its absence or delay past 6 months of age is suggestive of mental retardation,³ an autism spectrum disorder, or other developmental disorder (see Zwaigenbaum et al., 2005).

Delayed Language Development

From the behavior sample in the fi Albert's overall language development indicates clear delays compared to an average child his age. Most infants whimper and cry from moments after birth, and Albert does demonstrate both on the fi Watson and Rayner (1920) noted nine instances in which Albert whimpered and/or cried during their study. At several points during the (silent) fi Albert makes a "cry face," which may indicate concurrent crying.

Other than his ability to whimper and cry, however, Albert falls far behind the timeline for the average infant. Normally, gurgling and cooing is observed between 1 and 3 months of age, random babbling at about 4 months of age, repetitions like "ba ba ba" or "da da da" by 6 months, and run-on multiconsonant streams (e.g., "ba ba, ga ga, da da, ba ba") at 9 months (O'Grady, Archibald, Aronoff, & Rees-Miller, 2005). By 9 months of age, babbling begins to be associated with correlated, rhythmic arm movements, as part of the emergence of an integrated speech-gesture system (Iverson & Fagan, 2004).

What does Albert exhibit? Because the fi is silent, I could not conclusively assess Albert's verbalizations, but there is no frame in which he appears to move his lips to speak or babble. Neither does Albert gesture with his arms and hands at any point in the fi. We only have two notations from Watson and Rayner (1920) of any near-verbalization by Albert. The fi instance occurs following the eighth aversive trial with the rat, when Albert is 11 months and 20 days old:

[He] fell over to the left side, got up on all fours and started to crawl away. On this occasion there was no crying, but strange to say, as he started away he began to gurgle and coo, even while leaning far over to the left side to avoid the rat. (Watson & Rayner, 1920, p. 7)

This sequence begins at frame 20100 (11 min, 10 s into the fi). Albert's face is out of view for much of the scene. When his face is visible, no

articulatory movements are apparent. Albert, nearly a year old and under aversive stimulation, is gurgling-cooing. Gurgling-cooing would be age-normal for a 1- to 3-month-old child, and found almost exclusively in a social, affectional context (O'Grady et al., 2005).

The second instance of vocalization reported by Watson and Rayner (1920) occurred when Albert was 12 months and 21 days of age. Albert has the rabbit placed in front of him, and "then as the rabbit came nearer he began pulling his feet away, nodding his head, and wailing 'da da'" (Watson & Rayner, 1920, p. 11). Under conditions of aversive stimulation and escape responding, gurgling and cooing at 11 months and 20 days, and a single wailed "da da" utterance at 1 year, 21 days of age (when he should be chattering and already possess a several word vocabulary beyond "mama"), suggest developmental delay and/or neurological immaturity (Feldman & Messick, 2008).

Lack of Social Referencing

In many respects, the Watson and Rayner (1920) study is a prototype of the triadic arrangement used in later studies of infant social referencing (e.g., Campos & Stenberg, 1981). In these investigations, infants with caretakers positioned off to the side are confronted with strangers, visual cliffs, strange toys, or animals. By 9 months, the vast majority of infants visually "consult" their caretakers when shown strange objects (Feinman, Roberts, Hsieh, Sawyer, & Swanson, 1992). Never, during any of the filmed stimulus presentations, does Albert display social referencing.

Typically, 8-month-olds exhibit a key building block of social referencing: "gaze monitoring," that is, following the caretaker's gaze as a guide to what is important to notice (Klinnert, Campos, Sorce, Emde, & Svejda, 1983). Gaze monitoring is thought to be an important "joint attention milestone" in the development of social awareness (Johnson & Myers, 2008). Not once does Albert exhibit any indication of gaze monitoring. Instead, Albert's gaze appears almost entirely "stimulus bound," focused strictly upon the object before him. Significant absence or delay in markers of joint attention, such as gaze monitoring, suggests neuropsychological compromise in frontal executive functions (see Posner & Rothbart, 2007), forcing a number of

differential diagnoses, including an autism spectrum disorder (Johnson & Myers, 2008).

Rarely does Albert look away from the stimulus directly in front of him. Two or three times, he does briefly gaze blankly in the general direction of the camera. It is unclear if he is responding to a visual stimulus, orienting toward an auditory cue, or simply moving his head. In none of these cases does he show a brow flit to indicate recognition; Albert does not smile, nor does he appear to be gaze tracking.

Only twice during the film does Albert appear to make eye contact with either Watson or Rayner. The first instance (frame 18133; 10 min, 5 s into the film) is after Watson has presented the monkey to Albert and is about to show the dog. The baby twists around to his left with his left arm flailing and appears to look at Rayner's face. Because of Albert's head position, eye contact cannot be verified.

Why would Albert orient toward Rayner when Watson is presenting him with novel animals? During this film segment, Albert exhibits no obvious withdrawal responses. Rayner, however, appears to be touching Albert's back and may be providing a tactile distracter. A splice, which occurs at this point in the film, further complicates the interpretation of this possible interaction. Two white pads under Albert suddenly disappear following the splice. We do not know what occurred in the interim.

The second time that Albert may have made eye contact is following the presentation of the rabbit: "After a few seconds he puckered up his face, began to nod his head and to look intently at the experimenter" (Watson & Rayner, 1920, p. 11). This moment is captured on film at frame 19421 (10 min, 47 s into the film) and lasts 0.5 s. Watson is not visible during the scene, so it cannot be determined if Albert is seeking Watson's gaze or orienting to his voice. No evidence is provided of mutual gaze, or that Albert sees Watson or is responding to any of Watson's specific actions.

Albert's Environment and the Context of Filming

Could Albert's temperamental, social, and communicative deficits have been due to psychosocial deprivation? Severe abuse or neglect can cause infant speech delays and abnormalities (Leung & Kao, 1999), as can early nonex-

posure to language (Jones, 1995). Similarly, findings from Ceausescu-regime Romanian orphanages suggest that severe infant social deprivation can produce a “quasi-autistic” syndrome with language and communication deficits (Hoksbergen, ter Laak, Rijk, van Dijkum, & Stoutjesdijk, 2005; Rutter et al., 1999).

These cases, however, represent extremes of neglect and do not resemble the social conditions at the HLH. Attending physicians, residents, and interns rounded frequently, observing and interacting with patients. Albert’s mother was a wet nurse; she most likely had intimate contact with her son. Albert’s level of care almost certainly surpassed the minimal conditions necessary for the development of normal patterns of responding and social interaction.

It is also unlikely that some aspect of the filming context, such as frigid temperatures or bright lights, is responsible for Albert’s deficits. Albert’s attire when he was filmed at 8 months and 26 days of age suggests that the laboratory was not cold. He is shirtless and his vest is unbuttoned, allowing his ample belly to protrude. Some infants may have been exposed to the winter weather as they were transported to Watson’s laboratory, but that would not be a concern for Albert. He lived in the HLH (Watson & Rayner, 1920), Watson’s laboratory was in the Phipps Clinic, and the two adjacent buildings were connected by a corridor.

Was Albert “blinded” by bright lights used in the film? Certainly babies will avert their gaze to avoid bright lights. But bright lights, even if they were present, could not account for many of Albert’s age-delayed behaviors (e.g., his amimia, deficits in social referencing, and language deficits). We cannot measure the intensity of lighting that may have been employed, but there is some evidence that Watson recognized that, in some segments of the film, low rather than bright illumination was a problem. In one of the film scenes of *The Experimental Investigation of Babies*, a young woman to Watson’s left points a flashlight at another baby being tested for the palmar grasp reflex. This flashlight is probably an amateurish and ineffective attempt to augment the level of illumination. Two months later, when Albert is 11 months old, he is in a different room with better lighting. We cannot establish with certainty why Watson chose to conduct the latter phases of the study in a different environment, but the quality of illumination may have been a factor.

Summary and Implications of Behavioral Analysis

Albert’s temperament and behavior are not within the normal range for his age, and the abnormalities observed on film cannot solely be attributed to the hospital environment or the physical context of filming. Numerous diagnoses suggest themselves, including mental retardation, an autism spectrum disorder, or another pervasive developmental disorder. Differential diagnosis from Albert’s on-film behavior is impossible posthumously, since it would require specialized behavioral, genetic, and/or neuropsychological testing (Semrud-Clikeman & Elison, 2007; Wolraich et al., 2008).

There is also the possibility that Albert’s deficits were not due to attentional failures per se. Because all stimuli were presented directly before Albert, defects in visual perception or eye tracking may have caused him to be inattentive to any but the most salient visual stimuli. Indeed, Albert’s two strongest reactions were to the clang of the bar and the bark of the dog (Watson & Rayner, 1920). Neither the sight of the dog, burning newspaper inches from his face, nor any other visual stimulus evoked reactions of comparable intensity. A careful reading of Watson and Rayner’s (1920) description of Albert’s first confrontation with the white rat also suggests that the baby was visually impaired:

He allowed the rat to crawl toward him without withdrawing. He sat very still and fixated it intently. Rat then touched his hand. Albert withdrew it immediately, then leaned back as far as possible but did not cry. When the rat was placed on his arm he withdrew his body and began to fret, nodding his head. The rat was then allowed to crawl against his chest. He began to fret and then covered his eyes with both hands. (p. 11)

Sitting still and fixating intently while being approached by a fear-arousing stimulus, and then reeling at its first touch, is inconsistent with phobic avoidance. It is, however, consistent with defective visual perception. It may be telling that Watson and Rayner (1920) repeatedly noted Albert’s preference for wooden blocks as playthings throughout the study, and the blocks were used to quiet him throughout the trials. For a child with defective vision, blocks may have been favored in part because they were static with sharply defined edges, and thus were more easily seen and handled. Albert’s affinity for the blocks would also be consistent with the pref-

erence for “hard” objects over soft ones (e.g., blankets or stuffed animals), which is commonly observed in infants with autism spectrum disorders (Johnson & Myers, 2008).

If a child such as Albert came to my professional attention, I would refer him to a pediatric neurologist to rule out any acute systemic illness or neuropathology that might account for his unresponsiveness. A host of congenital, chronic neurodegenerative diseases can masquerade as developmental syndromes and would need ongoing medical management. More critically, acute conditions like infectious, metabolic, or toxic encephalopathies could produce behavior like Albert’s, are sometimes reversible, and would require immediate medical attention.

Regrettably, I could not refer Albert to a pediatric neurologist; I could only refer his video. I was fortunate to have as a colleague William Goldie, a pediatric neurologist with a 30-year clinical, teaching, and research career, and specialties in infant arousal, hypotonia, and facial diplegias such as Moebius syndrome. Coincidentally, Goldie did his pediatrics residency at the Johns Hopkins HLH. After 90 years, Albert would once more be examined by a Johns Hopkins physician.

Neurological Analysis (Fridlund, Goldie)

In mid-August 2010, I contacted Goldie and explained that I had historical film footage of a 9-month-old “whose cognitive and neurological status was uncertain.” When he first viewed the film, Goldie was unaware that he was assessing Little Albert. At several points, he asked me to play and replay portions of the video. I took notes while Goldie provided commentary:

He’s obese . . . a chubby little kid who doesn’t move around very much . . . Too passive . . . He grabbed past the pencil . . . He scooped at the block . . . he’s raking-scooping instead of pincer grasping . . . He’s not at a 9-month level . . . He’s not looking at the person in the fire scene . . . He’s not showing fear to the monkey . . . [He does not] show the “anxiety-curiosity complex” . . . no approach-avoidance ambivalence . . . He’s so unaware of the people around him.

Three things bother me: a lack of social awareness . . . very primitive scooping, normally there’s pincer mid-line play by 8 months, by 9 months they’re usually very dexterous, developmentally he’s less than 8 months. . . What bothers me the most, a lack of anxiety mixed with curiosity; there is no startle to animals.⁴

I asked Goldie if these signs might be due to psychosocial deprivation. He replied that the infant in the film resembled babies with plagiocephaly (flat spots on the head), who were often described as “too good.” These babies were neurologically compromised to begin with, but their apparent complacency often resulted in a lack of caretaker attention. As we viewed more footage, Goldie continued to comment, “Lack of scanning in his eyes . . . Blank face . . . masked facies [facial appearance].”

At this point, I opened the “blind,” revealing that the boy in the film was Little Albert. I also told Goldie there was reason to believe that the child died with hydrocephalus. I asked whether such a child, if referred to him, would lead him to a specific neurological diagnosis. Goldie was convinced that Albert had some kind of abnormality, but stated, “[His condition could be] anything . . . there are two things about Albert. I’d be worried about autism, and I’d be worried about retardation . . . [there’s] some underlying defect . . . possibly leukodystrophy [a deterioration of myelin in the brain].” After watching the video, Goldie concluded, “There’s something already gone wrong.”

Goldie’s assessment supported my analysis. The behavioral and neurological evidence strongly suggests that Albert’s brain functioning was compromised at the time Watson filmed him.⁵

Evaluations of Albert on Film: A Summary (Fridlund, Goldie)

Our video analyses led to overall clinical impressions of Albert that, from psychological and neurological vantage points, overlapped considerably.

From a Psychological Perspective

The “stolid and unemotional” temperament described by Watson and Rayner (1920) understates the filmed presentation of Little Albert (Watson, 1923), which is one of passivity, unresponsiveness, amimia, lack of social awareness and social referencing, and stimulus-boundedness. Together, these features suggest mental retardation and/or a number of other developmental disorders. Many of Albert’s reactions are also consistent with defects in visual perception.

From a Neurological Perspective

Goldie's evaluation, conducted blindly and independently of Fridlund's prior behavioral assessment, revealed an immaturity that is unexplained by the psychosocial environment. Instead, Albert's movements and responses suggest neurological abnormality. These include passivity, hand-scooping in lieu of pincer-grasp movements, masked facies, lack of eye scanning and social awareness, and the absent or deficient approach-avoidance response complex when confronted by strange objects. Together, these features suggest diagnostic alternatives that include mental retardation, an autism spectrum disorder, and several other congenital neurological disorders.

If Albert B. is Douglas Merritte, then he probably suffered from a congenital or early onset neurological condition (such as congenital hydrocephalus), or perhaps a neurodegenerative disease, that accounts for the entire set of impairments seen on film and his eventual death at the age of 6 years.

Douglas Merritte's Family History (Fridlund, Irons)

I sent my notes to Beck, who forwarded them to Gary Irons. I was eager to speak with Mr. Irons to hear firsthand about his uncle Douglas and grandmother Arvilla. We spoke for nearly an hour on September 15, 2010. Our wide-ranging discussion initially covered much of the information reported by Beck et al. (2009). Although I have been primed by clinical experience and knowledge of "false memory" research to discount the value of recollection (Bjorklund, 2000), I was struck by the detail in Irons's recollections and the care with which he distinguished what was uncertain from what he knew "for sure."

I asked Irons what specific things, if any, he might have heard about Douglas, especially regarding his physical condition. He responded without hesitation: "I do know that he was never able to walk." I was stunned and asked him to elaborate:

She [Arvilla] had to carry him from room to room. I think he could crawl. I heard it from my mother who was the same age as Douglas. The two families were friends, and I assume that my mother might have played with him when they were young. Apparently,

he would . . . I don't know if he could talk . . . he would indicate he wanted to go from one room to another, then Arvilla would have to pick him up.

I told Irons that I regarded this information as extremely significant. Failure to walk likely excluded autism and a range of communicative disorders as diagnoses. We discussed what kinds of disorders might have affected Douglas, and I pledged to Irons that I would keep him informed.

That same day, I e-mailed Goldie and relayed Irons's recollections. Goldie noted that numerous neurodegenerative diseases could account for such a presentation, including inborn metabolic errors or leukodystrophies such as Krabbe Disease. He concluded, "We may never be able to find the definite and definitive diagnosis."

By late September 2010, the video analyses were mostly concluded, and I (Fridlund) sent a working draft to Beck for comments. The infant's deficits were glaring, and Goldie and I had come to similar conclusions. Nonetheless, we recognized that making posthumous, phenotypic psychological and neurological assessments from experimenter notes and old film clips was an imprecise endeavor. In addition, the discrepancy between the death certificate and Irons family history regarding the onset of Douglas's hydrocephalus had not been resolved. We needed additional medical documentation to validate our psychological and neurological evaluations, and to determine if Douglas had been hydrocephalic prior to 1922.

The Missing Key: Douglas Merritte's Medical Records (Beck, Fridlund, Irons)

While gathering material for the Beck et al. (2009) article, Beck asked several times to see the HLH patient and/or employee records for the years 1919–1920. He was told that these files no longer existed (A. Harrison, personal communication, August 6, 2008). Surprisingly, we later learned that some HLH medical records survived (P. E. Letocha, personal communication, May 6, 2010).

As Douglas Merritte's next of kin, Gary Irons applied to Johns Hopkins to gain access to his uncle's medical files. In early October 2010 (G. Irons, personal communication, October 1, 2010), a parcel of loose pages—copies of physician's and nursing notes—arrived at Irons's door. On a personal basis, Douglas's medical

history furnished members of the Irons family with much-needed information about their genetic background.

Irons allowed Fridlund, Beck, and Goldie to review the notes, and worked with his coauthors to integrate Douglas's medical data with Irons family lore.⁶ From a historical perspective, Douglas's medical records provided the validity criterion we were seeking. A close parallel between the analyses of Albert on film and Douglas's medical files would not be found unless (a) Fridlund's and Goldie's evaluations were basically accurate, (b) the onset of Douglas's hydrocephalus was prior to 1922, and (c) Douglas was Little Albert.

In addition to furnishing a means to validate our previous findings, Douglas's medical documents presented an intriguing opportunity. By concurrently examining multiple information sources, we could derive new findings that would be unobtainable from any single source. For example, if we assume that Douglas is Albert, we could now discover the calendar dates upon which Watson and Rayner (1920) performed their assessments. This would only require adding Albert's age at each session (from Watson & Rayner, 1920) to Douglas's date of birth (Department of Health and Mental Hygiene, 1919).

Of course, combining information from both Douglas and Albert is only warranted if they are the same person. Fortunately, the interactions of the various data types themselves provide a test of the appropriateness of this procedure. If they are the same person, the key dates for Albert should dovetail temporally with those for Douglas. For example, Albert's test dates should not occur when Douglas is in surgery. We proceeded to examine this chronology.

Converging Data Sources: A Chronology Emerges (Fridlund, Beck, Irons)

Douglas and Arvilla: March to November 1919

Douglas was born on March 9, 1919; the birth was "normal." Mother and child were discharged from the obstetrical department at Johns Hopkins on March 20 (Merritte Medical Files [MMF], March 20, 1919).⁷ Douglas's condition was noted as "excellent." A brief family history taken at the time referred to Douglas as

an "illegitimate child" and listed Arvilla's residence as the "Exeter Street Home," another name for the Baltimore Home for Fallen and Friendless Women (MMF, March 20, 1919).

It soon became apparent that Douglas was not the healthy child that he appeared to be at birth. He was readmitted to the HLH on Saturday April 19, at the age of 6 weeks. Findings included a "staring expression" and an anterior fontanelle that was "widely open." Douglas's eyes protruded "slightly" and tended "to be rotated downward." His reflexes were "markedly hyperactive everywhere"; his arms were "flexed and trembling continuously and legs flexed at its thighs and knees." Projectile vomiting occurred shortly after admission and Douglas was "restless" and cried "constantly" (all from MMF, April 19, 1919).

On April 20, a puncture was made into the lateral ventricles of Douglas's brain (done with a needle through the soft spot of the skull). The fluid was cultured and was clear of infection. Phenolphthalein dye was injected into the ventricle, which produced "considerable reaction" (probably an anaphylactic reaction due to phenolphthalein hypersensitivity; see International Agency for Research on Cancer, 2000; Kendall, 1954), but "no stimulants" such as adrenalin (i.e., epinephrine) were deemed necessary to resuscitate him. A second puncture into the lumbar (lower back) region of the spine followed, and the cerebrospinal fluid (CSF) was assayed to determine the presence or absence of the dye (MMF, April 20, 1920).

No dye was found in Douglas's lumbar fluids, which confirmed a diagnosis of obstructive (noncommunicating) hydrocephalus, a blockage of normal flow of CSF that can result from congenital anomalies, tumors, cysts, and so forth. By 1915, the use of paired punctures with ventricular dye infusion and lumbar dye assay was a standard procedure for the differential diagnosis of obstructive hydrocephalus (Frazier, 1915). On Monday April 21, the day following Douglas's diagnosis, Arvilla Merritte was "engaged as a wet nurse" at the HLH (MMF, April 21, 1919).

Also in accordance with the standard of practice at the time (Zingher, 1919), over the next several months, Douglas received a series of additional ventricular and lumbar punctures to relieve the excessive, continually accumulating CSF. Ventriculo-peritoneal shunts for infants

were not available in 1919, nor were neurosurgeries available for resolving infant ventricular defects. On May 1, Douglas received the second of such paired punctures with dye administration and was found to have a “temp elevated slightly” (MMF, May 1, 1919).

On May 4, 1919, Douglas received his third set of punctures. He was found to have contracted streptococcal meningitis and ventriculitis, as confirmed by cultures of his CSF. The infection was treated with injections of antineurococcocal serum. Also on May 4, an injection of phenolphthalein appears to have induced an other toxic reaction, with “feeble rapid pulse, irregular shallow breathing, stupor, and moderate cyanosis.” Adrenalin was administered and “recovery was quite prompt” (MMF, May 4, 1919).

During his stay at the HLH, Douglas received a total of 9 paired ventricular and lumbar punctures, performed on April 20, May 1, May 4, May 5, May 8, May 10, May 16, June 9, and June 28, with phenolphthalein given on April 20, May 1, May 4, May 5, May 16, and June 9. No dye was injected on the dates of May 8 or May 10, and its use on June 28 is uncertain (MMF, respective dates).

Much of May and June of 1919 was concerned with managing Douglas’s high fevers, which often exceeded 100 and peaked at 103 degrees (MMF, May 4, 1919). For several days during this febrile episode, his head was severely retracted (MMF, May 8, 1919). Both the high fever and head retraction resolved by mid June, as Douglas recovered from the meningitis/ventriculitis (MMF, June 9, 1919; June 12, 1919). Although the infections responded to treatment, projectile vomiting continued and an unexplained lowgrade fever persisted during much of this period (MMF, June 24, 1919).

The June 24 entry also reported that Douglas “cries loudly frequently” (MMF, June 24, 1919). This was consistent with previous entries; frequent crying was noted on April 19, April 20, and April 27. The medical records make no mention of Douglas crying after June 24, presenting the possibility that his disposition may have changed as a result of further neurological deterioration. Thus, Watson’s (1930) report that “we never saw him cry until after our experiments were made!” (p. 159) may accurately reflect Douglas Merritt’s temperament when he was tested.

Following the June 28 puncture procedure, Douglas became critically ill. Although parts of the record are almost illegible, those notes that can be deciphered depict a severe anaphylactic reaction: “The child acted strangely and seemed to have become suddenly limp,” “Patient is spastic, with priapism almost constantly” (priapism is a prolonged penile erection that sometimes accompanies spasticity), “apparently unconscious, several times up to 2 ½ minutes without breathing . . . then the heart would become inaudible . . . interrupted by intense rapid panting respirations, during which the cyanosis that had become intense would quickly disappear.” The episode appears to have prompted the administration of adrenalin, atropine, and morphine (all from MMF, June 28, 1919). Convulsions and priapism required at least another day to resolve (MMF, June 29, 1919). Punctures were suspended for the remainder of his initial admission to the HLH.

Throughout most of July, August, and September, Douglas gained weight and his condition was “steadily improving.” A setback occurred in October when he “contacted dysentery.” By midNovember, Douglas had recovered; his “temperature returned to normal on 11/10/19 and has remained normal since” (MMF, November 15, 1919). An examination on November 24 (MMF, November 24, 1919) found that Douglas’s head circumference was 46.5 cm, referred to an 8month norm of 44.0 cm. Enlargement of the head and bulging of the anterior fontanelle, controlled initially by the repeated punctures, were recorded throughout the remainder of his stay at the HLH.

Watson and Rayner: March to November 1919

From March until June 1919, Watson was teaching at Johns Hopkins and chairing the Psychology Department. Rosalie Rayner was completing her final year at Vassar and graduated on June 10 (D. M. Rogers, personal communication, September 30, 2008). We do not know Rayner’s location during the summer of 1919, but a letter from Watson (1919a) to Bertrand Russell reveals that he left Baltimore on June 6 to vacation with his family in Ontario.

The preface for *Psychology from the Standpoint of a Behaviorist* (Watson, 1919f) is dated September 1919. Unlike later editions, which

extensively covered the Albert case (e.g., Watson, 1924), the first edition contained no mention of the infant. Watson returned to Baltimore shortly before classes resumed on September 30 (University Register, 1919–1920). At that time, Rosalie Rayner began her first semester as a graduate student in Watson’s laboratory.

Throughout October and November, Watson (1919c, 1919e) sought funds to purchase fish. On November 19, 1919, President Goodnow (Watson, 1919d) of Johns Hopkins obtained approval for Watson to acquire 1,000 ft (304.8 m) of fish stock.

Four Lives Converge: Douglas, Arvilla, Watson, and Rayner—November 1919 to October 1920

Douglas’s health appears to have temporarily stabilized at the time Watson and Rayner were preparing for their conditioning study. A brief entry in early December stated that Douglas had gained one pound during the previous week and that his “feedings were well taken” (MMF, December 1, 1920). If Douglas is Albert and Watson and Rayner (1920) accurately reported the infant’s age on the various test dates, Albert’s baseline data were collected and fish was given on December 5.⁸

On December 6, an X-ray of Douglas showed “very marked hydrocephalus” (MMF, December 6, 1919). The X-ray fish was not included in the records sent to Mr. Irons. We do not know if it survives in the John Hopkins medical archives.

No acute illnesses were reported in December, although projectile vomiting and one “elevation of temperature” were observed (MMF, December 14 and 22, 1920, respectively). Following repeated concern about the child’s vision, examination by the eminent ophthalmologist Leo J. Goldbach in late December (at age 9 1/2 months) revealed optic nerve atrophy with thinning of the retinal choroid (MMF, December 20, 1919).

The University closed for Christmas vacation from December 24 through January 4 (“University Register, 1919–1920,” 1919). According to a census taken on January 2, 1920 (U.S. Bureau of the Census, 1920), Arvilla was living on the Johns Hopkins campus; she listed her profession as “foster mother.” There is no evidence that Douglas or Arvilla left Johns Hopkins during the holiday season. The vacation may, how-

ever, have delayed filming the conditioning sequences.

Douglas was diagnosed with the measles on January 24, 1920; the eruption was “about at height” on January 27, and by January 29, his temperature had returned to normal (MMF, respective dates). Nothing in the medical documents suggests that measles, a disease that causes great distress in many children, produced crying or other signs of great discomfort in Douglas. Instead, Douglas, who cried loudly and frequently earlier in his infancy, exhibited a reaction that was only “somewhat fretful” (MMF, January 24, 1920).

Watson and Rayner’s (1920) second test session was conducted when the child was 11 months, 3 days old. For Douglas, this would have meant a date of February 12, 1920, 2 weeks after his recovery from the measles. A mid-February entry from Douglas’s records states that “from a nutritional point of view, the child is doing very nicely. He vomits occasionally, as he had been doing” (MMF, February 14, 1919).

Albert’s ages on the third through the sixth test dates were 11 months, 10 days; 11 months, 15 days; 11 months, 20 days; and 12 months, 21 days. Adding the ages to Douglas’s date of birth shows that these sessions were held on February 19, February 24, February 29, and March 30, 1920. No emotional evaluations were performed between the penultimate and final assessments, but “Albert was brought weekly to the laboratory for tests upon right and left-handedness, imitation, general development, and so forth,” (Watson & Rayner, 1920, p. 10).

Although Douglas’s head continued to enlarge, he contacted no acute illnesses and was generally stable on each of the session dates. Neither was he in surgery, recovering from a recent procedure, nor undergoing diagnostic tests that would have precluded him from participating in the Watson and Rayner (1920) investigation. Douglas was scheduled to be discharged from the HLH on March 31, possibly to coincide with the end of his mother’s employment (MMF, March 24, 1920). For unknown reasons, Arvilla went against medical advice and removed Douglas from the ward on March 24.

We were unable to ascertain Douglas’s and Arvilla’s exact whereabouts during the spring and summer of 1920, but, almost certainly, they

were in the Baltimore area.⁹ On August 27, 1920, Douglas was “brought back for an operation” and presented with extremities that were “somewhat spastic.” At 17 months, his head circumference of 53 cm placed him above the 98th percentile for his age. There seemed to be a progressive decline in visual perception; it was observed that “the pupils are large but react well to light” but “he takes no notice of objects” (all from MMF, August 27, 1920).

Additional ventricular and lumbar punctures were performed, again with the injection of phenolphthalein. This reconfirmed the diagnosis of obstructive (noncommunicating) hydrocephalus (MMF, August 27, 1920). By the next morning, Douglas was running a temperature of 99 degrees, which progressed to 100 degrees by the evening. Repeat punctures were performed the next day (MMF, August 28, 1920). Following another introduction of the dye on August 28, Douglas began running sustained high fevers that peaked at 105 degrees on August 29. His temperature began to fall by September 2. During this period, he remained “very spastic” (MMF, September 2, 1920). Arvilla removed Douglas from the HLH on September 5; his condition was listed as “unchanged” (MMF, September 5, 1920).

The summer of 1920 was a difficult one for Watson (for a detailed description of Watson’s intellectual environment and trajectory at Johns Hopkins, see Leys, 1984). An extramarital affair with Rayner became public, leading to the collapse of his marriage. Rayner left Baltimore in May (Watson, 1920a), but her absence did not prevent a scandal. Watson’s position became increasingly untenable as the summer progressed, and he was forced to resign from the Johns Hopkins faculty in the first week of October (Watson, 1920b).

What Does Douglas Merritte’s Medical History Imply About Little Albert on Film? (Goldie, Fridlund)

Douglas’s medical records establish definitively that his hydrocephalus was congenital; its onset was not in 1922, as Dr. Crunk reported on the death certificate. The medical documents confirm Gary Irons’s understanding that Douglas “always had problems,” but they ended any hope that Douglas may have had a few healthy years before his early death. Although Douglas

recovered from the acute ailments that so plagued his infancy, he would always be impaired.

Infants so ill from a combination of congenital hydrocephalus, meningitis, and ventriculitis are likely to suffer many forms of brain damage. Hydrocephalus alone can produce brain damage, due to compression lesions from high CSF pressures. Infections of the brain can cause severe damage to brain structures and scarring both in and around the brain. In addition, streptococcal infection can produce toxic reactions that result in permanent brain damage. Common findings in children who have suffered from these conditions include diminished reactivity and hypofrontality. Albert’s diminished reactivity was marked throughout the video; bilateral frontal lobe dysfunction is evident in Albert’s amimia, as well as a notable facial hypotonia (lowered muscle tone), manifest in his droopy eyes, puffy cheeks, and draping of the mouth.

Douglas was confirmed to have optic nerve damage, also a likely result of the combination of pressure, inflammation, and toxicity. The record contains several references to defects in visual perception and insensitivity to surroundings, but importantly, at 17 months, he still had pupillary responses to light (MMF, August 27, 1920). This indicates that Douglas was not completely blind but retained at least partial retinal function, and that the connections to his pretectal brain stem nuclei were intact. It is likely that Douglas incurred damage that produced visual defects commonly associated with hydrocephalus, such as oculomotor nerve deficits (e.g., double or blurred vision), or perceptual defects related to damage higher in the nervous system (e.g., difficulty in attending to and apprehending objects, i.e., apperceptive agnosia; see, e.g., Milner, 1995), which would produce behavior much like Albert exhibited on film.

Congenital noncommunicating hydrocephalus has many possible causes, including arteriovenous malformations, cysts, and malignant tumors such as astrocytomas. These primary lesions may progress and have independent effects of their own (Avellino, 2005). Intrauterine infections, hemorrhages, and toxins can cause scarring of the cerebral aqueduct that usually drains CSF from the ventricles; rare X-linked genetic disorders are also associated with malformations that can produce inadequate drainage (Sarco et al., 2008). Such kinds of aqueductal stenosis (stricture), if com-

plete, can result in subsequent noncommunicating hydrocephalus.

We cannot know what primary obstructive defect caused Douglas's hydrocephalus, but eventual failure to walk, as Irons recalled, and convulsions as a cause of death, as Crunk recorded, are consistent with brain damage that was a result of the medical complications he suffered during his first year of life. He was clearly never medically normal and was severely ill from the age of 6 weeks to 1 year of age, during which time he remained within the HLH.

The signs and symptoms of any neurological disorder differ on a case-by-case basis, and this is especially true in complex cases such as Douglas Merritte's, in which a primary lesion produced hydrocephalus, which was then complicated by meningitis and ventriculitis, along with several acute allergic reactions to injected dye. The combination of a chronic, progressive neurological condition and multiple life-threatening febrile illnesses can have diffuse, widespread, deteriorative effects upon the developing brain.

Additionally, drowsiness and lethargy often accompany the progression of congenital hydrocephalus (Avellino, 2005). It is instructive that Douglas, a baby who had been observed to cry frequently, was last noted to cry on June 24, just after the resolution of his prolonged febrile episode (MMF, June 24, 1919). This diminished postfebrile responsiveness probably contributed to Albert's being, in Watson and Rayner's (1920) terms, "stolid and unemotional" (1920, p. 1).

Two other aspects of Douglas's case must be mentioned. The first concerns the repeated administration of phenolphthalein dye with his punctures. Douglas had a total of four acute episodes—three of which were arguably life-threatening—that may have been hypersensitivity reactions to phenolphthalein (MMF, April 20, May 4, and August 28, 1920; the notes for the June 28, 1919, episode are illegible, in part, and we are uncertain whether phenolphthalein was injected). The medical rationale for such repetitions after the initial diagnosis of obstructive hydrocephalus, especially given Douglas's febrile toxic reaction, is unclear and worrisome.

The second aspect is the fact that Douglas developed both streptococcal meningitis and ventriculitis while he was at the HLH (bacterial cultures obtained at his first set of punctures just

after admission were negative; MMF, April 20, 1919). The former is routinely contagious, but the latter would require introduction of the infection into Douglas's ventricular space. According to the medical records, the "patient was admitted to the hospital on April 19, 1919, with a diagnosis of hydrocephalus. Tests showed that it was of the internal obstructive type. In making the tests a streptococcus nonhemolytic meningitis was caused" (MMF, August 28, 1920). This is frank admission that the near-lethal infection that so devastated Douglas's early development and, we believe, diminished his responsiveness, was iatrogenic. We have not been able to determine the exact nature of this iatrogenic causation; presumably, the infection "was caused" accidentally (e.g., via improper needle sterilization), but we cannot exclude the possibility that the causation was experimental (i.e., Douglas may have been used for research by investigators other than Watson).

In addition to providing a chronology of Douglas's life at the HLH, his medical documents furnished a criterion upon which to validate our evaluations of Albert on film. Suffice it to say, all the anomalies that we observed in our analyses of Albert are consistent, and commensurate, with the degree of neurological impairment we discovered later in the medical records of Douglas Merritte (see Figure 1).

Douglas Merritte and Albert B.: Interlocked Chronology and Pathology (Beck)

The preceding chronology, assembled by combining information pertaining to Douglas with information pertaining to Albert, is justification only if they are the same infant. This logic extends to the day-to-day changes in Douglas's medical status and the dates that Watson tested Albert. If Douglas is Albert, then the session dates, as calculated from Douglas's birth and Albert's age at each testing, should fall on those days in which it was possible to perform the study.

The time frame for our analysis is bounded by Douglas's birth date, March 9, 1919, and the approximate date of Watson's dismissal from Hopkins, October 4, 1920, a total of 576 days. These 576 days can be viewed as a product of days on which it was possible to collect data and days on which data collection was impossible. In determining whether data could be gathered,

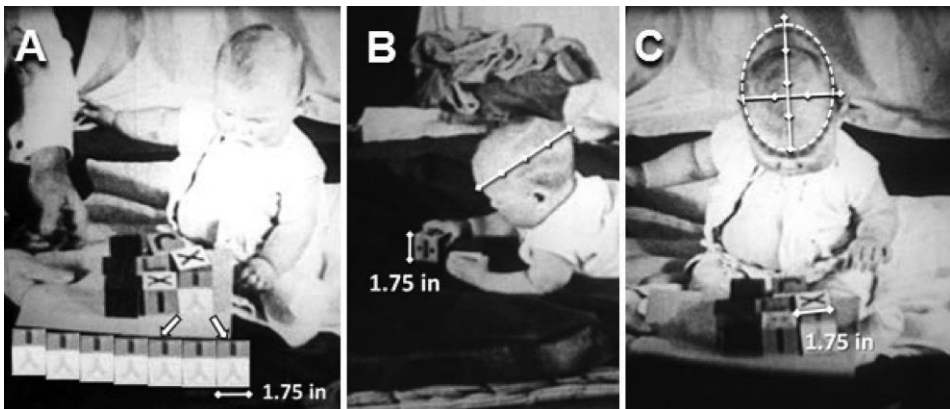


Figure 1. Photogrammetric estimation of Albert’s head circumference. Apart from the facial similarities between Albert B. and Douglas Merritte reported by Beck et al. (2009), the two had strikingly similar skull circumferences. Fridlund estimated Albert’s head circumference from the Watson (1923) film by using the wood blocks Watson gave to Albert as a standard metric, and modeling his head as an ellipse with short and long axes measured from film frames containing frontal and profile views of Albert. (A) Frame 12100 (6 min, 44 s from the film’s beginning) shows Watson presenting Albert a set of wooden blocks atop a stiff piece of paper; the block near and parallel to paper’s edge was digitally cloned and inserted *seriatim* underneath the paper to reveal a block edge width of 1.75 in (a standard for blocks at the time) that matches $(6.3 \text{ widths} \times 1.75 \text{ in per width} = 11.025 \text{ in})$ the long edge of standard 8.5 X 11-in paper. (B) Profile view provided by frame 13591 (7 min, 33 s from the beginning), which allowed using the 1.75-in block at Albert’s midline as a metric to derive a frontal-to-occipital distance for Albert of 3.8 block widths, or 6.65 in. (C) Middle block positioned below crest of Albert’s head was used as 1.75-in metric to estimate Albert’s head width, suggesting an approximate head width of 2.8 block widths, or 4.9 in. Dashed ellipse is drawn with skull length obtained in B; using the average of the long and short diameters provided by views in B and C, the resulting ellipse (the product of π and the average of the diameters converted to cm) yields a minimal circumference for Albert of approximately 46.1 cm at 8 months and 26 days of age. Records from The Johns Hopkins University medical archives indicate that Douglas Merritte’s head circumference was 46.5 cm at 8 months, 14 days of age (MMF, November 24, 1919). Thus, the predicted head circumference of Albert estimated by the elliptical model differed from the measured head circumference of Douglas Merritte by less than 1% at ages 15 days apart. This provides further corroboration of the identity of Little Albert as Douglas Merritte. Further details of the analysis and its assumptions and limitations can be provided upon request.

it is necessary to take into consideration that (a) the baseline was filmed and (b) Watson, Rayner, and Albert appeared in the baseline and conditioning segments of the movie. Therefore, data could not be collected if any of the following held true: (a) money had not yet been appropriated for the film (256 days), (b) Watson was outside the Baltimore area (approximately 110 days), (c) Rayner was known to be outside the Baltimore area (approximately 236 days), or (d) Douglas was in surgery, was undergoing extensive tests, or was acutely ill (approximately 69 days).

If we apply this algorithm, we find approximately 407 days (71%) in which data could not

have been collected. Only 169 of the original 576 days remain as possible test days (29%). Albert was assessed by Watson and Rayner (1920) approximately nine times (it is unclear how many “weekly” evaluations were conducted between the penultimate and final session). Each of the nine investigative sessions falls on a possible test date. The events mesh without conflict, providing support for combining documents referring to Douglas with those referring to Albert.¹⁰

Likewise, the psychological and neurological analyses of Albert B. on film are entirely consistent with an infant with the medical history of Douglas Merritte. Taken together, the medical

records, Watson's (1923) film, Watson and Rayner's (1920) report, Watson's correspondence, and Irons family history yield a detailed account of the infant's stay at the HLH. As we shall see, a concurrent assessment of multiple information sources also brings to light information that Watson may have considered in deciding when to test Albert.

A Summary of the Evidence: Was Albert a Neurologically Damaged Child? (Beck, Fridlund)

The initial objective of this inquiry was to examine Watson and Rayner's (1920) claim that "Albert's life was normal: he was healthy from birth and one of the best developed youngsters ever brought to the hospital" (p. 1). That contention cannot be reconciled with the facts that we have uncovered about Douglas Merritte. The data strongly support the proposition that Douglas/Albert was a neurologically damaged child at the time he was tested by Watson and Rayner (1920).

The counterargument to our conclusion is that Albert B. was healthy and he cannot have been Douglas Merritte. In taking this position, one must dispose of (a) consistencies in the evaluations of Albert on film and Douglas's medical documents, (b) commonalities in the histories of Albert B. and Douglas Merritte (residence, birth, maternal occupation, physical appearance, etc.) reported by Beck et al. (2009), (c) the overlap between Douglas's availability for testing and Albert B.'s session dates, (d) the head circumference correspondence between Albert B. and Douglas Merritte (see Figure 1), and (e) the absence of an alternative candidate meeting even a subset of the criteria.

Implications of the Findings for Watson's Legacy (All Authors)

Several issues are compelled by these findings. The first question to consider is why Watson and Rayner's (1920) claim that Albert was a "healthy" and "normal" infant was accepted for so long, despite clear evidence to the contrary. Other issues are methodological—they further limit our confidence that any conditioning of Albert occurred. The final two issues are more troublesome. They concern the misrepre-

sentation of Albert/Douglas's medical condition and the propriety of inducing fear in a neurologically impaired baby.

Why Were Albert's Abnormalities Ignored?

Thousands of educators and parents at public speaking engagements by Watson in the 1920s and 1930s, students in psychology classes throughout the twentieth and into the twenty-first centuries, and now viewers on YouTube and Google Video have watched film segments of Watson's purportedly "healthy" and "normal" infant, Albert B. No doubt, many health professionals have seen Watson's (1923) movie. Some must have noticed that Albert was unresponsive and "slow," even if no one publicly made the leap to neurological impairment.

One reason that Little Albert's deficits may have been overlooked is the powerful lock that expectations have on perceptions, a finding that goes back to Leeper (1935). Until faced with information to the contrary, most scientists assume that fellow investigators have made a good faith effort to portray their data accurately. Watson repeatedly restated the "normal" and "healthy" character of Albert in his writings and lectures, and as we have seen, his description readily metastasized to secondary sources. Like so many paired-associate learning trials, the "Albert/healthy" theme took hold and became axiomatic. Watson and Rayner's (1920) most effective conditioning may not have been of Albert but of their readership. Watching Little Albert with the stipulation that he was "healthy" and "normal" made it easy to overlook the infant's deficits.

Implications of Albert's Health for His "Conditioning"

Many observers have noted that the Watson and Rayner (1920) study is a flawed demonstration of Pavlovian fear conditioning (for relevant issues, see Bouton & Moody, 2004; Colwill & Rescorla, 1986; Rescorla, 1988). As summarized by Harris (1979), these shortcomings include (a) little evidence for any kind of lasting conditioning, much less a phobia; (b) Watson's frequent striking of the metal bar when Albert reached out to touch the stimulus, a procedure more like operant punishment than classical

conditioning; (c) the use of only one participant; (d) employing measurements of Albert's fear that were wholly subjective; and (e) multiple failures to replicate the results.

To these methodological flaws, we must now add the overly long time interval between Albert's "baseline" and the first conditioning session. Because more than two months passed between baseline and the first conditioning session, any changes in Albert's responses might have been due to age rather than acquired fears. Watson and Rayner's (1920) procedure confounded fear acquisition with maturation.¹¹

Why would Watson, a competent experimentalist, employ an obviously defective design? Watson and Rayner (1920) provided no rationale and we cannot account for the entire 2-month delay. Christmas holidays and Douglas's health, however, may have been contributing factors.

Johns Hopkins was on vacation from December 24, 1919, through January 4, 1920 ("University Register, 1919–1920," 1919), and there were a number of days on which it would have been difficult or impossible to test Douglas. He was X-rayed (MMF, December 6, 1919), given otoscopic (MMF, December 22, 1919) and ophthalmoscopic (MMF, January 20, 1920) examinations, and had fevers for 2 days and measles for 5 days (MMF, December 14, 1919; December 22, 1919; MMF, entries for January 24 through January 28, 1920). On January 29, there was "a case of diphtheria on the ward" (MMF, January 29, 1920). Douglas did not contract the disease, but the outbreak may have made Watson reluctant to bring the infant to his laboratory. We cannot overlook the possibility that personal matters may have absorbed Watson's time and served to extend the interval between baseline and conditioning.

Another puzzling design issue is why Watson selected a neurologically compromised infant, specifici Douglas, to condition. At first glance, a "normal" baby would be the logical choice. Presumably, a more cognitively developed child would be easier to condition and the results would have greater generality. According to Watson and Rayner (1920), Albert was chosen because he was "stolid and unemotional" (p. 1) and would experience "relatively little harm" (p. 2) from the fear induction procedure. If we accept the investigators' rationale,

a concern for children prompted them to select such an impassive baby.

The selection of Douglas/Albert also allowed Watson and Rayner (1920) to meet a number of experimental exigencies. Watson's first requirement was that the participant must behave differently during the pre- and postconditioning sessions. This necessitated finding an infant who would yield a very low level of responding during baseline. Many babies would not have reacted passively when large animals and fire were thrust before them. Douglas's stolidity and/or inability to perceive stimuli insured that Watson obtained the low baseline he desired.

Watson and Rayner (1920) also needed a baby who would be available for an extended period, to assess the "effect of time upon conditioned emotional responses" (p. 10). Arvilla's occupation and/or her son's health would have made it clear to Watson that Douglas was likely to remain at the HLH for a prolonged stay. Finally, Watson needed to locate a mother who would not or could not deny his request to induce fear in her child. We have no objective data, but it seems improbable that most parents in the 1920s would have permitted curious investigators to scare their children. Douglas's mother was not just another parent. She was a wet nurse employed by the HLH.

Wet nurses were generally held in disrepute as "fallen women," and while their life-giving mother's milk was prized (infant mortality was far lower among breast-fed than bottle-fed babies; Golden, 2001), they were devalued. At the turn of the century, a professor of pediatrics demeaned them as "one part cow and nine parts devil" (cited in Golden, 2001, p. 155). Watson knew that Arvilla was a wet nurse and may have taken her social status into account when selecting Douglas. Had Douglas been the son of more socially prominent parents (e.g., a banker's child), it seems unlikely that he would have been subjected to the fear induction procedure.

Arvilla's position was further compromised because she worked and lived at Johns Hopkins. Most importantly, Douglas was receiving expensive medical care that she could not afford. Although we have no specific knowledge of Douglas's case, illegitimate infants with such an illness history were frequently offered up as "experimental material," with minimal scrutiny and few protections (see Lederer, 1984, 2003). Such a combination of factors would have left

Arvilla ill disposed to refuse a request from Watson or a Johns Hopkins physician to experiment on her child. Voluntary consent, as we understand the term today, was not possible to give or to withhold.

Did Watson Know He was Conditioning a Neurologically Damaged Child?

The graver issues raised by this study concern Watson and Rayner's (1920) contention that "Albert's life was normal: he was healthy from birth and one of the best developed youngsters ever brought to the hospital" (p. 1). What did Watson know when he wrote those lines? Did he realize that Douglas/Albert was neurologically impaired?

In Watson's defense, it should be noted that he is not mentioned in Douglas's medical files. Neither were we able to locate a document in which Watson or Rayner referred to Albert's poor physical condition. If such a record existed, it was probably destroyed. Late in life, Watson burned his papers, declaring, "When you're dead, you're all dead" (Buckley, 1989, p. 182).

We cannot establish with absolute certainty that Watson knowingly misrepresented Albert's condition. We can, however, show that he had access to many information sources, any one of which would have led him to realize that he was inducing fear in a very impaired child. First, there are Watson's own observations. The investigators assessed Albert at least nine times over an interval of almost 4 months. Watson presented himself in 1920, as he would years later, as an authority on infant behavior. With his supposed expertise, he should have easily noted Albert's masked facies, lack of gaze tracking, absent vocalization and gesturing, immature hand-scooping, and visual deficits. Indeed, much of *The Experimental Investigation of Babies* (Watson, 1923) shows Watson and Rayner performing tests of infant reflexes that are used to verify neurological intactness.

Even if Watson failed to recognize that Douglas/Albert was not a normal infant, it is difficult to believe that everyone at Johns Hopkins was equally oblivious to the child's condition. Albert lived in the HLH, a mecca for pediatric neurology. By 1908, Harvey Cushing, at Johns Hopkins, had attempted a venous shunt for infant hydrocephalus (Pendleton, Zaidi,

Jallo, Cohen-Gadol, & Quiñones-Hinojosa, 2010), and by 1913, his student Walter Dandy had begun the research that defines modern hydrocephalus typology and treatment (Dandy & Blackfan, 1913). Almost certainly, an infant exhibiting the abnormalities that Albert displayed on film would have been noticed and treated.

Once Albert was diagnosed, the small size of the campus would have almost insured that Watson knew the infant's condition. The Phipps Clinic, which housed Watson's laboratory, was adjacent to the HLH. It strains credulity to suggest that no one would have mentioned Albert's health problems, especially since Watson and Rayner (1920) report that their initial impressions of Albert's responses "were confirmed by the casual observations of the mother and hospital attendants" (p. 2).

If Albert received medical care, then his treatment would have been recorded. Watson presumably had access to these records. Are we to believe that Watson was so negligent an investigator that he never consulted the medical files of the child featured in his film?

If, as our findings suggest, Douglas is Little Albert, then it is almost inconceivable that Watson was unaware of the baby's medical status. Douglas lived in a ward for very sick children, a fact that Watson or Rayner must have realized when they or a staff member transported the child to the Phipps Clinic laboratory. Finally, it is possible, but extremely unlikely, that the nine session dates, calculated by adding Albert's age to Douglas's birthday, fell by chance on days when Douglas's condition was relatively stable and Watson and Rayner were available to assess the infant. A more reasonable explanation is that the dates mesh because Watson took the infant's health into consideration when deciding when to test him.

To make the argument that Watson did not know he was inducing fear in a neurologically impaired baby, it is necessary to contend that (a) the correlation between Douglas's health and the test sessions is happenstance, (b) Watson did not bother to check the medical files, (c) no one told Watson that Albert was chronically ill and had several serious acute episodes, and (d) Watson failed to notice abnormalities in the infant he tested repeatedly. The evidence is circumstantial, but the data strongly support the premise that Watson knowingly misrepresented Albert's medical condition.

Watson's Conduct in the Little Albert Study (All Authors)

Today, intentionally misreporting scientific information and producing fear in a neurologically damaged child might result in academic dismissal, professional censure, and civil and criminal charges.¹² Of course, applying twenty-first-century standards to a study conducted in 1920 would be inappropriate and unfair—a classic instance of presentism. A more useful frame of reference is to consider how the misreporting of data, and the instilling of fear in a neurologically impaired infant, would have been viewed by Watson's contemporaries.

The Misrepresentation of Medical Information

In addressing this question, it is important to distinguish between the investigators' descriptions of Albert's reactions to the test stimuli and their account of the infant's health. We have no evidence that the researchers inaccurately described the delivery of the stimuli or Albert's responses to them. With some minor exceptions, the filmed segments of Albert correspond to the written report (Watson & Rayner, 1920).

It is possible that Watson believed that the details of the conditioning procedure were important but that the health and the identity of the child were inconsequential. If so, he was entirely mistaken. Knowing Albert's medical condition alters our interpretation of his behavior and his reactions to the experimental stimuli. As a violation of the norm of faithful and complete reportage in science, such an omission would compromise "certification" of the knowledge (Zuckerman, 1977) and render replication impossible. It would be impermissible in the science of his, or any, time. Incomplete or unfaithful reportage was one of the more common offenses that Charles Babbage cited in his renowned rant against the malpractices of the Royal Society (Babbage, 1830).

Although we cannot know Watson's exact motivations, we can identify several benefits he gained in presenting Albert as a healthy child. Certifying Albert's excellent development and phlegmatic disposition shielded Watson from charges of maltreatment of children (see Hackling, 1991, for the evolution of current conceptions of child abuse). Although the American

Psychological Association (1953) did not pass a formal code of ethics until 1953, Watson recognized that inducing fear in infants was controversial (e.g., Watson, 1928). Proffering Albert's emotional stability assured readers that he would suffer "relatively little harm" (Watson & Rayner, 1920, p. 2) and deflected possible criticism from the investigators and Johns Hopkins University. Albert also needed to be "healthy" and "normal" for Watson to advance his aim of establishing general laws of learning.

Inducing Fear in a Neurologically Damaged Child

There is now general agreement that, because of their greater vulnerability, mentally retarded and developmentally disabled individuals require special protections from experimental abuse (see discussions of issues by Melton & Stanley, 1996; Ross, 2004; and Weisstub & Arboleda-Flórez, 1997). That was not the case in Watson's time. Mentally retarded and developmentally disabled children were among the "available and contained" populations (after Reverby, 2011) most used for medical research.

Institutionalized children, whether orphaned or disabled, were especially prized because they could be used to study human disease under controlled conditions (Ross, 2004). As Lederer (2003) ably documented, institutionalized children were used to test a host of early vaccines, including those for syphilis, tuberculosis, and diphtheria. These tests often required the direct injection of the suspect pathogens to prove causation.

Research using institutionalized children extended to invasive surgical procedures. For example, in 1896, Arthur Wentworth performed lumbar punctures on over 45 sick infants at Boston Children's Hospital for procedural reasons only (Lederer, 1984). Hines Roberts studied the CSF of newborns by performing spinal punctures on 423 African American babies in an Atlanta hospital (Roberts, 1925, cited in Lederer, 2003). These studies provoked outcries from human antivivisectionists (who were allied with the animal antivivisectionists of the day) and consternation among some medical researchers, but no censure or any official ethical stand on human experimentation was taken (Lederer, 1984). By comparison, Watson's conditioning

procedures with Albert were mild and noninvasive.

The proponents of medical experimentation on children used the promise of future treatments and cures to deflect scrutiny and allay criticism of their procedures. As the acclaimed virologist Thomas Rivers, who trained at Johns Hopkins, was on its medical staff from 1919–1922, and later headed the Rockefeller Institute for nearly 20 years, admitted, “Well, all I can say is, it’s against the law to do many things, but the law winks when a reputable man wants to do a scientific experiment . . . Unless the law winks occasionally, you have no progress in medicine” (cited in Reverby, 2011).

Watson was no less promissory about his psychological procedures or certain of their justification. In the Albert study, he aimed to show that a “normal” infant could be made to have arbitrary, strong, and possibly permanent emotional reactions to random objects, using just a few simple Pavlovian trials. In 1930, he posed his famous challenge, after a Jesuit maxim, to “give me a dozen healthy infants well-formed, and my own specified world to bring them up in and I’ll guarantee to take any one at random and train him to become any type of specialist I might select—doctor, lawyer, artist, merchant-chief and, yes, even beggar-man and thief, regardless of his talents, pendants, tendencies, abilities, vocations, and race of his ancestors” (Watson, 1930, p. 82). If psychology could do that, the possibilities would be limitless—the wholesale positive transformation of society through scientific child rearing, including the control of the emotions.

Watson (1928) rejected charges that he was mistreating his participants, arguing, like the medical experimentalists surrounding him, that the ends justify the means:

You may think that such experiments [referring to the Albert study] are cruel, but they are not cruel if they help us to understand the fear life [sic] of the millions of people around us and give us practical help in bringing up our children more nearly free from fears than we ourselves have been brought up. They will be worth all they cost if through them we can find a method which will help us remove fear. (p. 54)

Decades would pass before medicine and psychology adopted ethical standards for research on children and other vulnerable populations, and this kind of consequentialist, cost-

benefit calculus was repudiated ethically and legally (see Beecher, 1966; Grodin & Glantz, 1999; and Ross, 2004, for reviews).

Douglas Merritte died 5 years after the Watson and Rayner (1920) study, one of thousands of anonymous “experimental children” whom science and the law failed to protect. Irons family lore has it that Douglas never walked and may never have learned to speak. His hydrocephalus progressed until his death by convulsions.

John B. Watson, however, gifted Douglas with immortality. He made Douglas psychology’s legendary “lost boy.” Advertising himself as an expert on child development, Watson developed the cover story that Douglas/Albert was “healthy” and “normal,” and used the “Little Albert” study as one of the bases for the best-selling *Psychological Care of the Infant and Child* (1928), which preached regimentation and stoicism over spontaneity and nurturance, and profoundly influenced the ways of child rearing for generations to come.

Endnotes

1. The photogrammetric analysis of Albert’s head from the cinematic footage bears on Albert’s diagnosis and identity, and, due to length restrictions, is depicted and summarized in Figure 1.
2. Filming was conducted in the winter of 1919–1920, most likely using 35-mm stock, the most popular format of that era. The earliest existing version of Watson’s (1923) film is in 16 mm, a format that was introduced that year by Eastman Kodak (Borum, 2004). Technicians, probably associated with the Stoelting Company, converted the original film to the newer format. The most common versions of both formats used a 24-frames-per-second (fps) frame rate. This frame rate was sufficient to capture the adults, babies, and animals in Watson’s film at normal speed, with none of the jerkiness often found in earlier movies, or, as they were then called, “flicks.” The DVD capture up-sampled the film to a 30-fps frame rate, preserving all the information on the 16-mm film.
3. We use the term “mental retardation” throughout this paper without prejudice, in accordance with current diagnostic terminology for individuals with cognitive deficits. For the upcoming *DSM-5*, the Neurodevelopmental Disorders Work Group intends to substitute “Intellectual Disabilities” for the current term, which many consider pejorative (American Psychiatric Association, 2009).

4. Watson and Rayner (1920) recorded Albert as demonstrating such ambivalence much later than age-normal, at the final testing session when he was 1 year and 21 days of age. When presented the fur coat, “again there was the strife between withdrawal and the tendency to manipulate” (p. 10). This final session was not filmed.
5. For concurrent validity, we sought a third assessment by a clinical expert who, like Goldie, was blind to the hypothesis. Accordingly, Fridlund obtained a consultation with Professor Jill Waterman, Department of Psychology, University of California, Los Angeles (J. Waterman, personal communication, September 1, 2011). Waterman, a specialist in childhood psychopathology, is Coordinator of the UCLA Psychology Clinic, and Director of the Infant Mental Health program at UCLA TIES for Families. (None of the authors had prior acquaintance with her.) We first asked her just to offer informal clinical impressions as she examined the video of Albert (she did not recognize “Albert” during the interview). While viewing Albert at 8 months, 26 days, her observations included, “His reaching isn’t as focused as you’d expect for a 9-month-old . . . He doesn’t have as good a grasp as you’d expect . . . The crawling looks a little delayed . . . He is less reactive to both the film and the dog than you’d expect . . . Sometimes he holds his hands a little unusually, like you’d see in a less mature child . . . He doesn’t have smiling or affective responding, he doesn’t really cry and he doesn’t really smile . . . There’s something about his face that’s different . . . His expression doesn’t ever really change.” When she viewed the video of Albert at 11 months, Waterman noted, “He doesn’t seek comfort . . . normally you’d think the child would turn to the caregivers . . . I don’t think he’s as reactive to all these negative stimuli as other babies, he’s definitely not able to use these people to turn to for support . . . You’d think he might turn to that woman for solace for help to regulate his emotions, even if he didn’t know her . . . I would expect more turning for help.” After viewing both video segments, I asked if, mindful that 4 min of video could never be diagnostic, she had any tentative hypotheses or diagnostic impressions, and she offered, “There’s mainly a question about responsiveness. He isn’t really moving all that much. The most striking thing is that when he’s distressed, he doesn’t know what to do. If I had to think of two hypotheses regarding this child’s behavior, they would be (1) severe deprivation, neglect, or abuse (this could account for the “frozen” responsiveness), or (2) some kind of neurological or developmental-delay syndrome (I’m thinking of the unusual face, and the fine motor delay).” Thus, Waterman’s observations substantially corroborate our (Fridlund’s and Goldie’s) notations of Albert’s anmia, lack of social referencing, poor grasp, and overall diminished responsivity, and she detected the neurological involvement in Albert’s condition.
6. We are sensitive to the privacy issues involved in releasing protected health information (PHI). Mr. Gary Irons, Douglas Merritte’s next of kin, is the holder of the privilege for the PHI contained in his uncle’s medical records. As coauthor, he has read and approved for publication every sentence in this article. Many of the documents referenced in this article are housed at the Johns Hopkins University or the Archives of the History of American Psychology. Qualified scholars may make application to review these and many other papers related to Watson.
7. Hereafter, we merely use the MMF acronym and the date of entry to refer to the chart entries contained within the Merritte Medical File, which denotes the collection of loose copies of papers released as PHI (see note 6) to Irons, and, in turn, provided to his coauthors on this paper (G. Irons, personal communication, October 1, 2010).
8. Powell (2011) recently voiced concern about the timeline and Albert’s age at the baseline and first filming, and suggested that it vitiates the identification of Albert as Douglas Merritte. Relying on a letter written by Watson (1919b), Powell proposed that the first assessment of Albert might have occurred after December 5, 1919. Powell’s contention is neither consistent with the information presented in this report, nor does it accord with information supplied by Watson and Rayner (1920). Acceptance of his proposition requires that we assume that Watson and Rayner (1920) either misreported the age of Albert at testing or the number of days separating the penultimate and final sessions.
 In his many descriptions of the study, Watson (e.g., Watson & Rayner, 1920; Watson & Watson, 1921) tells us that (a) Albert was first assessed at 8 months and 26 days of age, (b) he aged 1 month and 1 day between the next-to-last (11 months, 20 days of age) and last (12 months, 21 days of age) session, and (c) 30 days separated the penultimate and final sessions.
 Some complex Gregorian arithmetic proves necessary and decisive. Albert could only have aged 1 month and 1 day in a 30-day period if the penultimate assessment occurred during a month with 29 days. To illustrate, one can locate Feb-

ruary 20 during a leap year and count forward 30 days. The thirtieth day is March 21. Now, one can add 30 days, assuming that penultimate session was in a month with 28 days, 30 days, and 31 days. The calculations demonstrate that in 30 days, the infant aged 1 month and 2 days, 1 month and 0 days, and less than 1 month, respectively. Thus, if Watson and Rayner's (1920) reporting is accurate, the next-to-last session must have been during February 1920, a leap year.

The last day in which Albert could have aged 1 month and 1 day in 30 days is February 29, 1920. Subtracting 11 months and 20 days (Albert's age at the penultimate session) from that date reveals that he was not born after March 9, 1920. Adding 8 months and 26 days (the age at first testing) to March 9 reveals that the initial assessment was no later than December 5, 1919. Powell's proposal that the first filming was performed after December 5 would have the next-to-last session no earlier than March 1, 1920, a month with 31 days. If the penultimate assessment was in March, Albert would have aged less than one month in 30 days.

My students and I (Beck) recognized that the first session almost certainly was on or before December 5, long before we knew of Douglas Merritte. Nonetheless, we (Beck et al., 2009) did not wish to completely eliminate the possibility of a later filming because, as Powell (2011) notes quite correctly, there are discrepancies in Watson's many reports of the Albert study. Our practice is to accept any uncorroborated statement by Watson only with reservation.

It is not difficult to identify examples of Watson's careless reporting. Although he usually indicates that the final two sessions were 30 days apart (e.g., Watson & Rayner, 1920, p. 13; Watson & Watson, 1921, p. 514), in at least one instance he states that the separation was 31 days (Watson & Rayner, 1920, p. 10). If the child aged 1 month and 1 day in 31 days, then the penultimate session must have been in April, the final session in May, and the initial filming in January. These dates are much later than Powell (2011) or any other scholar has proposed for the Albert study.

Douglas Merritte's birth date dovetails with Albert's ages at testing and the days between the last two test sessions. Douglas was 8 months and 26 days of age on December 5, 1919, the outside date of what we had previously considered the likely range for the first session. We are not aware that anyone has disputed Beck et al.'s (2009) contentions that no more than four wet nurses were concurrently in residence at the HLH, that Douglas was the son of a HLH wet

nurse, and that he lived at Johns Hopkins at the time of the Watson and Rayner (1920) study. Neither has anyone doubted that Douglas had a great deal in common with what is known of Albert.

If, as Powell suggests, the infant tested by Watson and Rayner (1920) was born after March 9, 1919, then Douglas could not be Albert. Powell is proposing a second baby in the nursery. That child would necessarily be very similar to Douglas. Like Douglas, Powell's hypothesized infant must be the son of a wet nurse, be Caucasian, have spent almost his entire first year at the HLH, and have left Johns Hopkins in the early 1920s. He must have also looked like Douglas (see biometric analysis reported by Beck et al., 2009) and have the same birth month. There are so many similarities that this hypothesized child could be Douglas's twin, except that they do not share the same date of birth.

We thank Powell for his interest and his thoughtful efforts to ascertain the exact date of the first filming. However, the proposition that the initial test session was after December 5, 1919, can only be maintained by arguing that (a) Watson and Rayner (1920) incorrectly reported Albert's age at testing or the total days between the penultimate and final sessions, (b) two remarkably similar infants were simultaneously in the same small nursery, and (c) the conclusions of this article are erroneous. Given the available evidence, it is highly improbable that the first test session was after December 5.

9. We have identified a number of locations where Arvilla and Douglas may have spent the spring and summer. After his initial discharge from the HLH, Douglas may have stayed for a short time with Arvilla in her room at Johns Hopkins. The pair might have returned to the Home for Fallen and Friendless Women or resided with two close friends in Baltimore. An attempted reconciliation with Douglas's father cannot be excluded. Gary Irons related a story of a possible family reunion, which occurred about the time Arvilla and Douglas left Johns Hopkins. When Arvilla moved to Baltimore, she left her older son, Maurice Irons, to be raised by his grandparents. Years later, Maurice told Gary that when he was a very young boy, he and his grandfather visited Arvilla in Maryland. What is known with certainty is that Arvilla and Douglas lived, for several years, with the Brashears family outside of Baltimore. As of yet, we have not been able to determine the exact date when they joined the Brashears household.
10. The dates used to make these calculations can be provided upon request.

11. A second technical issue arises with Watson's procedures. Fifteen years later, another breed of behaviorist, the contiguity theorist Edwin Guthrie, would demonstrate the need to consider not just whether an aversive stimulus worked, but what it impelled the organism to do. Watson wished to make Albert learn to recoil from a white rat, but did so by clanging a metal bar at Albert's back. When Watson clangs the bar, Albert falls forward instead, in the direction of the rat. Had Watson hidden the bar and struck it in front of Albert, the desired conditional response would have aligned with the unconditional escape response (see Guthrie, 1934, 1935).
12. We sidestep the issue of Rayner's culpability. Rayner was a graduate student under Watson's supervision, and so Watson bears major responsibility for the conduct and reporting of the study.

References

- American Psychiatric Association. (2009). *Report of the DSM-V Neurodevelopmental Disorders Work Group*. Retrieved from <http://www.psych.org/MainMenu/Research/DSMIV/DSMV/DSMRevisionActivities/DSM-V-Work-Group-Reports/Neurodevelopmental-Disorders-Work-Group-Report.aspx>
- American Psychological Association. (1953). *Ethical standards of psychologists*. Washington, DC: Author.
- Amiel-Tison, C., & Gosselin, J. (2001). *Neurological development from birth to six years* (C. Bastable, Trans.). Baltimore, MD: The Johns Hopkins University Press.
- Avellino, A. M. (2005). Hydrocephalus. In H. S. Singer, E. H. Kossoff, A. L. Hartman, & T. O. Crawford (Eds.), *Treatment of pediatric neurologic disorders* (pp. 25–36). Boca Raton, FL: Taylor & Francis.
- Babbage, C. (1830). *Reflections on the decline of science in England, and on some of its causes*. London, England: Fellowes.
- Beck, H. P. (2011). Finding Little Albert: A seven-year search for psychology's lost boy. *The Psychologist*, *24*, 392–395.
- Beck, H. P., Levinson, S., & Irons, G. (2009). Finding Little Albert: A journey to John B. Watson's infant laboratory. *American Psychologist*, *64*, 605–614. doi:10.1037/a0017234
- Beecher, H. K. (1966). Ethics and clinical research. *New England Journal of Medicine*, *274*, 1354–1360. doi:10.1056/NEJM196606162742405
- Bjorklund, D. F. (2000). *False-memory creation in children and adults*. New York, NY: Erlbaum.
- Bouton, M. E., & Moody, E. W. (2004). Memory processes in classical conditioning. *Neuroscience and Biobehavioral Reviews*, *28*, 663–674. doi:10.1016/j.neubiorev.2004.09.001
- Buckley, K. W. (1989). *Mechanical man: John Broadus Watson and the beginnings of behaviorism*. New York, NY: Guilford Press.
- Burum, S. H. (Ed.). (2004). *American Cinematographer Manual* (9th ed.). Hollywood, CA: ASC Press.
- Campos, J. L., & Stenberg, C. (1981). Perception, appraisal, and emotion: The onset of social referencing. In M. E. Lamb & L. R. Sherrod (Eds.), *Infant Social Cognition* (pp. 273–314). Hillsdale, NJ: Erlbaum.
- Cohen, M. E., & Duffler, P. K. (2003). *Weiner & Levitt's Pediatric Neurology* (4th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Colwill, R. M., & Rescorla, R. A. (1986). Associative structures in instrumental learning. In G. H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 20, pp. 55–104). New York, NY: Academic Press.
- Dandy, W. E., & Blackfan, K. D. (1913). An experimental and clinical study of internal hydrocephalus. *Journal of the American Medical Association*, *61*, 2216–2217.
- Department of Health, Bureau of Vital Statistics (Death Record Counties). (1925). Douglas Merritte, Carroll County, 10 May 1925 (MSA S1179, MdHR 50, 259–375, 2/56/62)[1]. Maryland State Archives, Annapolis, MD.
- Department of Health and Mental Hygiene, Division of Vital Records (Birth Record, BC). (1919). Baby Merritte, 70288, 02/25/04/006 (MSA T310–230). Maryland State Archives, Annapolis, MD.
- Feinman, S., Roberts, D., Hsieh, K.-F., Sawyer, D., & Swanson, D. (1992). A critical review of social referencing in infancy. In S. Feinman (Ed.), *Social referencing and the social construction of reality in infancy* (pp. 15–54). New York, NY: Plenum Press.
- Feist, J., & Feist, G. J. (2005). *Theories of personality*. New York, NY: McGraw-Hill.
- Feldman, H. M., & Messick, C. (2008). Assessment of language and speech. In M. L. Wolraich, P. H. Dworkin, D. D. Drotar, & E. C. Perrin (Eds.), *Developmental-behavioral pediatrics: Evidence and practice* (pp. 177–190). Philadelphia, PA: Elsevier Mosby.
- Frazier, C. H. (1915). Types of hydrocephalus: Their differentiation and treatment. *American Journal of Diseases of Children*, *11*, 96–102.
- Fridlund, A. J. (1994). *Human facial expression: An evolutionary view*. San Diego, CA: Academic Press.

- Golden, J. (2001). *A social history of wet nursing in America*. Columbus, OH: Ohio State University Press.
- Grodin, M. A., & Glantz, L. H. (Eds.). (1999). *Children as research subjects: Science, ethics, and law*. New York, NY: Oxford.
- Guthrie, E. (1934). Pavlov's theory of conditioning. *Psychological Review*, *39*, 199–206.
- Guthrie, E. (1935). The psychology of learning. New York: Harper & Row.
- Hacking, I. (1991). The making and molding of child abuse. *Critical Inquiry*, *17*, 253–288. doi:10.1086/448583
- Harris, B. (1979). Whatever happened to Little Albert? *American Psychologist*, *34*, 151–160. doi:10.1037/0003-066X.34.2.151
- Harris, B. (2011). Letting go of Little Albert: Disciplinary memory, history, and the uses of myth. *Journal of the History of the Behavioral Sciences*, *47*, 1–17. doi:10.1002/jhbs.20470
- Hoksbergen, R., ter Laak, J., Rijk, K., van Dijkum, C., & Stoutjesdijk, F. (2005). Post-institutional autistic syndrome in Romanian adoptees. *Journal of Autism and Developmental Disorders*, *35*, 615–623. doi:10.1007/s10803-005-0005-x
- International Agency for Research on Cancer. (2000). *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 71. Some antiviral and antineoplastic drugs, and other pharmaceutical agents* (pp. 387–415). Lyon, France: World Health Organization.
- Iverson, J. M., & Fagan, M. K. (2004). Infant vocal-motor coordination: Precursor to the gesture-speech system? *Child Development*, *75*, 1053–1066. doi:10.1111/j.1467-8624.2004.00725.x
- Johnson, C. P., & Myers, S. M. (2008). Autism spectrum disorders. In M. L. Wolraich, P. H. Dworkin, D. D. Drotar, & E. C. Perrin (Eds.). *Developmental-behavioral pediatrics: Evidence and practice* (pp. 519–577). Philadelphia, PA: Elsevier Mosby.
- Jones, P. E. (1995). Contradictions and unanswered questions in the Genie case: A fresh look at the linguistic evidence. *Language and Communication*, *15*, 261–280. doi:10.1016/0271-5309(95)00007-D
- Kagan, J. (2001). Temperamental contributions to affective and behavioral profiles in childhood. In S. G. Hofmann & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 216–234). Needham Heights, MA: Allyn & Bacon.
- Kassin, S. (2003). *Essentials of psychology*. New York, NY: Prentice Hall.
- Kendall, A. C. (1954). Fatal case of encephalitis after phenolphthalein ingestion. *British Medical Journal*, *1*, 1461–1462. doi:10.1136/bmj.2.4902.1461
- Klinnert, M. D., Campos, J. J., Sorce, J. F., Emde, R. N., & Svejda, M. (1983). Emotions as behavior regulators: Social referencing in infancy. In R. Plutchik & H. Kellerman (Eds.), *Emotions in early development: Vol. 2. The emotions* (pp. 57–86). New York, NY: Academic Press.
- Lederer, S. E. (1984). “The right and wrong of making experiments on human beings”: Udo J. Wile and syphilis. *Bulletin of the History of Medicine*, *58*, 380–397.
- Lederer, S. E. (2003). Children as Guinea pigs: Historical perspectives. *Accountability in Research*, *10*, 1–16. doi:10.1080/08989620300505
- Leeper, R. (1935). A study of a neglected portion of the field of learning—The development of sensory organization. *Journal of Genetic Psychology*, *46*, 41–75.
- Leung, A. K. C., & Kao, C. P. (1999). Evaluation and management of the child with speech delay. *American Family Physician*, *59*, 3121–3128.
- Leys, R. (1984). Meyer, Watson, and the dangers of behaviorism. *Journal of the History of the Behavioral Sciences*, *20*, 1984. doi:10.1002/1520-6696(198404)20:2<128::AID-JHBS2300200203>3.0.CO;2-J
- Melton, G. B., & Stanley, B. H. (1996). Research involving special populations. In B. Stanley, J. E. Sieber, & G. B. Nelton (Eds.), *Research ethics: A psychological approach* (pp. 177–202). Lincoln, NE: University of Nebraska Press.
- Milner, A. D. (1995). Cerebral correlates of visual awareness. *Neuropsychologia*, *33*, 1117–1130. doi:10.1016/0028-3932(95)00052-5
- O'Grady, W., Archibald, J., Aronoff, M., & Rees-Miller, J. (2005). *Contemporary Linguistics* (5th ed.). New York, NY: Bedford St. Martin's Press.
- Pendleton, C., Zaidi, H. A., Jallo, G., Cohen-Gadol, A. A., & Quiñones-Hinojosa, A. (2010). Harvey Cushing's use of a transplanted human vein to treat hydrocephalus in an infant in the early 1900s. *Journal of Neurosurgery: Pediatrics*, *5*, 423–427. doi:10.3171/2009.12.PEDS09388
- Pierce, W. D., & Cheney, C. D. (2004). *Behavior analysis and learning*. Mahwah, NJ: Psychology Press.
- Posner, M. I., & Rothbart, M. K. (2007). *Educating the human brain*. Washington, DC: American Psychological Association. doi:10.1037/11519-000
- Powell, R. A. (2011). Little Albert, lost or found: Further difficulties with the Douglas Merritte hypothesis. *History of Psychology*, *14*, 106–107. doi:10.1037/a0022471b
- Rescorla, R. A. (1988). Pavlovian conditioning: It's not what you think it is. *American Psychologist*, *43*, 151–160. doi:10.1037/0003-066X.43.3.151
- Reverby, S. M. (2011). “Normal exposure” and inoculation syphilis: A PHS “Tuskegee” doctor in

- Guatemala, 1946–1948. *Journal of Policy History*, 23, 6–28. doi:10.1017/S0898030610000291
- Roberts, M. H. (1925). The spinal fluid in the newborn with especial reference to intracranial hemorrhage. *Journal of the American Medical Association*, 85, 500–503.
- Ross, L. F. (2004). Children in medical research. *Perspectives in Biology and Medicine*, 47, 519–536. doi:10.1353/pbm.2004.0069
- Rutter, M., Andersen-Wood, L., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., . . . O'Connor, T. G. (1999). Quasi-autistic patterns following severe early global privation. English and Romanian Adoptees (ERA) Study Team. *Journal of Child Psychology & Psychiatry*, 40, 537–549. doi:10.1111/1469-7610.00472
- Sarco, D. P., Vanderbilt, D. L., & Riviello, J. J. (2008). Central nervous system disorders. In M. L. Wolraich, P. H. Dworkin, D. D. Drotar, & E. C. Perrin (Eds.), *Developmental-behavioral pediatrics: Evidence and practice* (pp. 359–383). Philadelphia, PA: Elsevier Mosby.
- Semrud-Clikeman, M., & Ellison, P. A. T. (2007). *Child Neuropsychology* (2nd ed.). New York, NY: Springer.
- Sroufe, L. A., & Waters, E. (1976). The ontogenesis of smiling and laughter: A perspective on the organization of development in infancy. *Psychological Review*, 83, 173–189. doi:10.1037/0033-295X.83.3.173
- Thompson, R. A., Easterbrooks, M. A., & Padilla-Walker, L. M. (2003). Social and emotional development in infancy. In R. M. Lerner, M. A. Easterbrooks, & J. Mistry (Eds.), *Handbook of Psychology. Vol. 6. Developmental Psychology*. Hoboken, NJ: Wiley.
- University Register, 1919–20. (1919, November). *Johns Hopkins University Circular*, 38 (9, Whole No. 319, New Series).
- U.S. Bureau of the Census. (1920). Johns Hopkins Hospital, Baltimore City, MD. In Fourteenth Census of the United States, 1920 (Enumeration District 82, Sheet 4A; Roll: T625_661). Retrieved June 29, 2009 from Ancestry Library database.
- Vander Zanden, J. W. (1981). *Human Development* (2nd ed.). New York, NY: Knopf.
- Watson, J. B. (1919a). *Letter to Bertrand Russell, October 4, 1919*. In the Cedric Larson Papers, Archives of the History of American Psychology, University of Akron, Akron, OH.
- Watson, J. B. (1919b). Letter to Frank J. Goodnow, December 5, 1919 (Record Group 02.001/Office of the President/Series 1/File 115, Department of Psychology, 1913–1919). Ferdinand Hamburger, Jr., Archives, The Johns Hopkins University, Baltimore, MD.
- Watson, J. B. (1919c). Letter to Frank J. Goodnow, November 13, 1919 (Record Group 02.001/Office of the President/Series 1/File 115, Department of Psychology, 1913–1919). Ferdinand Hamburger, Jr., Archives, The Johns Hopkins University, Baltimore, MD.
- Watson, J. B. (1919d). Letter to Frank J. Goodnow, November 17, 1919 (Record Group 02.001/Office of the President/Series 1/File 115, Department of Psychology, 1913–1919). Ferdinand Hamburger, Jr., Archives, The Johns Hopkins University, Baltimore, MD.
- Watson, J. B. (1919e). Letter to Frank J. Goodnow, October 27, 1919 (Record Group 02.001/Office of the President/Series 1/File 115, Department of Psychology, 1913–1919). Ferdinand Hamburger, Jr., Archives, The Johns Hopkins University, Baltimore, MD.
- Watson, J. B. (1919f). *Psychology from the standpoint of a behaviorist* (1st ed.). Philadelphia, PA: J. B. Lippincott. doi:10.1037/10016-000
- Watson, J. B. (1920a). Letter to Adolf Meyer, August 13, 1920 (Adolf Meyer Papers, Unit 1/3974/19), Alan Mason Chesney Medical Archives, The Johns Hopkins Medical Institutions, Baltimore, MD.
- Watson, J. B. (1920b). Letter to Frank J. Goodnow, October ?, 1920 (Office of the President/Series 1/File 115, Department of Psychology, 1920–1921). Ferdinand Hamburger, Jr., Archives, The Johns Hopkins University, Baltimore, MD.
- Watson, J. B. (Writer/Director). (1923). *Experimental investigation of babies* [Motion picture]. United States: C. H. Stoelting Co.
- Watson, J. B. (1924). *Psychology from the standpoint of a behaviorist* (2nd ed.). Philadelphia, PA: J. B. Lippincott.
- Watson, J. B. (1928). *Psychological care of the infant and child*. New York, NY: Norton.
- Watson, J. B. (1930). *Behaviorism* (rev. ed.). New York, NY: Norton.
- Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, 3, 1–14. doi:10.1037/h0069608
- Watson, J. B., & Watson, R. R. (1921). Studies in infant psychology. *Scientific Monthly*, 13, 493–515.
- Weisstub, D. N., & Arboleda-Flórez, J. (1997). Ethical research with the developmentally disabled. *Canadian Journal of Psychiatry*, 42, 492–496.
- Wolraich, M. L., Dworkin, P. H., Drotar, D. D., & Perrin, E. C. (Eds.). (2008). *Developmental-behavioral pediatrics: Evidence and practice*. Philadelphia, PA: Elsevier Mosby.
- Zingher, A. (1919). The value of ventricular puncture for the early diagnosis and serum treatment of posterior basilar meningitis. *American Journal of the Medical Sciences*, 157, 58–67. doi:10.1097/0000441-191901000-00008

Zuckerman, H. (1977). Deviant behavior and social control in science. In E. Sagarin (Ed.), *Deviance and social change* (pp. 87–138). Beverly Hills, CA: Sage.

Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life.

International Journal of Developmental Neuroscience, 23, 143–152. doi:10.1016/j.ijdevneu.2004.05.001