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Single Case Report

Life-long deficits in social adaptation and the frontal lobes: New evidence, seventy-five years after Ackerly and Benton's landmark case report of JP

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ABSTRACT

The case of JP, reported by Ackerly and Benton in 1948 with a detailed follow-up by Ackerly in 1964, stands as the index case of developmental prefrontal damage and its impact on social adaptation. Although the 1948 case report included findings from a 1933 pneumoencephalogram and exploratory craniotomy, a definitive cause was never established for JP's prefrontal damage. Etiologies were never determined for the left-sided seizures that occurred when JP was age four, nor for the progressive anterograde amnesia that JP developed in middle age.

Given Ackerly's thoroughness and long-term follow-up of his patient, it was hoped that a brain cutting would have been done, though no report of a post-mortem examination was published. The lead author of this paper (SB) set out to discover what had happened to

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JP after Ackerly's 1964 report and whether a brain cutting had in fact occurred. Using a variety of investigative approaches, it was discovered that a post-mortem brain examination had taken place. Those present at the brain cutting were identified, and the still-living witnesses to the brain cutting were interviewed. Previously unpublished, relevant materials were uncovered from archival sources. A film of the brain cutting, as well as photos, were located. A film of Ackerly interviewing JP prior to JP's death at age sixty-four also was found.

The authors studied autopsy findings in the newly discovered video and still images. These findings were judged consistent with massive perinatal hemorrhagic damage to both frontal lobes. JP's left-sided seizures were likely due to activation of a focus from his congenital brain damage. The anterograde amnesia that was documented when JP was twenty-five and that was noted to worsen when he was forty-nine remains unexplained but may have been related to slowly progressive hydrocephalus. This paper expands what is known about the case of JP, making it the only report of a person with congenital frontal injury followed for their entire life including post-mortem brain examination.

1. Introduction

In 1948, Drs. Spafford Ackerly and Arthur Benton, working at the University of Louisville, reported the case of JP, a young man they had first evaluated at age twenty in November 1933. Their patient had a history of childhood behavioral disturbances, including wandering and compulsively stealing automobiles. He had sustained massive early life damage to his prefrontal cortex (Ackerly & Benton, 1948). JP was the first well-studied case that demonstrated the impact of early life prefrontal damage on social adaptation and moral development.

Ackerly published additional details of JP's behavior in 1950 and 1964 (Ackerly, 1950, 1964). Benton published additional recollections of the case in 1991 and 2003 (Benton, 1991, 2003). Given Ackerly's detailed reporting and clear interest in following JP over time, the absence of a published account of JP's brain cutting and neuropathological findings was unexpected. One of the authors (SB) set out to discover whether JP's brain was studied post-mortem, what had become of JP's brain, and, if it could be located, whether behavioral–anatomical correlation might still be possible more than forty years after JP's death.

The search proved partially successful. A post-mortem brain cutting had, in fact, been done. Although the brain could not be located, living witnesses to the brain cutting were identified and a film of the event was found. The authors located and interviewed Ackerly's living family members and discovered archival material related to this case as well as a video recording of JP being interviewed by Ackerly in 1977.

In this report we summarize the published biographical and medical information about JP and discuss previously unreported information about the case, including JP's behavioral trajectory through to his death at age sixty-four. We interviewed physicians who participated in JP's care in his last year of life and reviewed newly discovered records from the period of 1975–1977. We also report our observations from the films of his brain cutting and Ackerly's 1977 interview with JP. Based on the photographic evidence, we were able to identify the most likely cause of JP's perinatal frontal damage and his seizures at age four, and contributing factors to the progressive anterograde amnesia he developed in adulthood.

2. Methods

In 1964, Ackerly wrote that "the last chapter in the history of our patient, JP, will not be written, of course, until later (Ackerly, 1964)." Based on this statement and on the thoroughness of Ackerly's 1964 report, we assumed that, if Ackerly knew of JP's death, a brain cutting would have taken place.

A search of the scientific literature yielded no reports by Ackerly about JP after 1964. However, an internet search uncovered an informal mention of a brain cutting of JP (Rice, 2007). In a phone interview, Dr. John F. Rice, the author of that report, identified the other witnesses to the post-mortem brain examination. Two of these individuals, Drs. Daniel Tucker and William Smock, were instructed by Ackerly to take photographs of the coronal sections and had retained copies of them.

We invited Ackerly's family members and the still-living witnesses to the brain examination to a meeting arranged at the University of Louisville Department of Psychiatry on June 17, 2017. Each participant was interviewed for their recollections of JP. A search of the personal papers of Spafford Ackerly in the Kornhauser Health Sciences Library Historical Collection at the University of Louisville uncovered additional documents related to Ackerly's care of JP.

One of the authors, Dr. Christine Adams, a psychiatry resident at the time of the 1977 brain cutting, provided a video of a case report on JP that she had presented at the 1978 American Psychiatric Association annual meeting (Adams Tucker, 1978). Her video contained a short clip of the post-mortem brain examination of JP. We now had evidence that a video of the brain examination had been made.

We located the complete film of the brain examination, digitized, and studied it. The authors also found and reviewed

records related to a 1977 psychiatric admission at age sixty-four; these included an admission summary, cranial computed tomographic images, a report of neurosurgical consultation, and an operative note documenting insertion of a ventriculoperitoneal shunt. Reports of psychological evaluations from 1975 and 1977 were reviewed as well as two letters written in 1936 from JP to his mother.

The case summary that follows is based both on the publications of Ackerly and Benton (Ackerly, 1950, 1964; Ackerly & Benton, 1948; Benton, 1991) and on the additional information uncovered by the authors.

Numerous attempts were made to locate any living relatives of JP. JP had no siblings and no descendants. Utilizing newspaper obituaries, cemetery and other public records, the authors constructed a family tree. We discovered no descendants of JP's parents' siblings using public records and social media outreach. The authors believe that JP has no living first or second-degree relatives. The Institutional Review Board of the University of Massachusetts T H Chan School of Medicine has determined that this study does not constitute human subjects' research.

3. Results

3.1. *Case report*

3.1.1. *Early childhood*

JP was born on December 29, 1912, in Louisville, KY, the 11.5-pound product of a relatively normal pregnancy and a difficult, 22-h labor. JP's mother had no known trauma during her pregnancy, however Ackerly did report that, while pregnant, she suffered from a vague illness and insomnia (Ackerly & Benton, 1948).

Based on the birth certificate we obtained, JP was delivered at home by Hugh Nelson Leavell. Dr. Leavell, who lived near JP's family, was a highly regarded pediatrician and generalist physician who also was regarded to be a skilled obstetrician (Editorial, 1924). The delivery required instruments and the mother suffered severe lacerations. The published reports did not describe any external signs of trauma to the infant that might have been related to the delivery. There were no neonatal convulsions. JP initially was unable to nurse, and by day nine he had lost 6.5 pounds and was jaundiced (Ackerly, 1964; Ackerly & Benton, 1948).

JP's development appeared to proceed normally. By the age of one year, JP began to walk and talk. However, by age two and a half he began to wander, sometimes a mile from home, without fear of being lost, and having no anxiety. On a few occasions at age three police needed to return him home.

Ackerly described the patient's father as a serious, moralistic, rigid, perfectionistic man. He worked as an automobile salesman and frequently traveled for business. The father often demanded adult behavior from young JP. He used corporal punishment for discipline, though this did not dissuade JP from wandering.

JP's mother had some college education. She felt her husband was too harsh with JP (Ackerly & Benton, 1948). Her own attitude toward her son was said to alternate between "overprotectiveness" and "irritability." Undeterred by parental discipline, JP continued to wander long distances, a habit that would persist into adulthood, and that JP once explained as "impulse" (Ackerly & Benton, 1948).

At age four, JP fell and hit his head on the floor while jumping on a bed. About an hour later he began to "act peculiarly," and to talk "like a smart alec." He lost consciousness and developed left-sided convulsive movements. He was taken to the hospital where, after four hours, the convulsive movements abated spontaneously, sparing him an exploratory craniotomy planned for that afternoon. By the time JP was discharged home the following morning, his neurological status had returned to baseline (Ackerly & Benton, 1948).

Throughout his childhood, JP exhibited stereotyped, excessively polite, courtly manners toward adults, described by Ackerly as "Chesterfieldian." This term alluded to the 18th century British statesman, Lord Chesterfield, known for his linguistic prowess and diplomatic skills. Even in childhood, JP's manners were regarded as shallow and superficial (Ackerly & Benton, 1948).

At school JP's behavior was both "Chesterfieldian" and incorrigible. His first-grade teacher was in the process of writing a letter to congratulate JP's parents on raising such a well-mannered child, when she looked up and discovered him exposing himself to the other children and masturbating (Ackerly, 1964). That year he also defecated in a paint bucket. In the second grade "he took a classmate's glove and rubber, defecated in them, and replaced the glove in the child's coat pocket (Ackerly & Benton, 1948)."

Later that year JP was caught exposing himself to two younger girls. When confronted, he denied it, saying, "I beg your pardon, sir!" He was subsequently transferred to a different school. This was the first of several school transfers that resulted from his problematic behaviors.

At the new school, JP passed the first three grades but had to repeat the 4th and 5th grades for disruptive behavior and poor attendance. He was described as an unpopular boy who was "boastful and bossy" toward younger children (Ackerly & Benton, 1948). "He would burst out in anger, and within a minute regain his composure and act as if his flare-up had never occurred. He would taunt a younger child for some seconds and then treat him with normal consideration (Benton, 1991)." Often, he was beaten up by classmates (Ackerly & Benton, 1948).

In 1926, at age thirteen JP was transferred to a "day school for defective children" as a consequence of numerous incidents of misbehavior, frequent reports of masturbating in school, and being caught stealing money from a girl at his school. This new day school referred JP to the Louisville Mental Hygiene Clinic for evaluation.

The intake evaluation at the Louisville Mental Hygiene Clinic indicated that JP lied, stole, and was known in his neighborhood for having "bad sex habits (Ackerly & Benton, 1948)." He lacked a sense of fairness and sportsmanship. He had no friends of either sex, did not remain long in any clubs he joined, and was never part of a gang. JP denied that he had fewer friends than other boys his age and, when confronted, attributed his own aberrant behaviors to others. He even claimed that it was other boys, not him, who engaged in public masturbation.

Academically, JP's teachers described his reading and spelling as good. His memory was very good, but his

arithmetic skills were poor (Ackerly, 1964). For entertainment, JP read books. According to his mother, he had excellent recall of the books' details. JP studied piano for two years, violin for a year, and ukulele for a few months. But he lacked sustained interest in any of his musical pursuits. He took dancing lessons for a year. He had "more than the usual" play equipment but, likely due to his habit of wandering, he was not given a bicycle. Ackerly commented that JP was not prone to daydreaming (Ackerly, 1964).

In 1926, the psychiatric evaluation in the Mental Hygiene Clinic concluded that JP was a "juvenile psychopath" who suffered from "parental mishandling (Ackerly & Benton, 1948)." Ackerly and Benton's paper indicate that JP's mother was largely blamed for his misbehavior. During a four-session trial of psychotherapy, JP showed only superficial intellectual understanding and agreed with the formulations of his therapist. His therapist concluded that the sessions had accomplished nothing (Ackerly & Benton, 1948).

JP was transferred from the day school for defective children to a small private school with individual tutors. He excelled academically. In 9th grade, he returned to public school, and was advanced to 10th grade because of his impressive language ability and his superficial good manners. Soon, however, he was demoted back to 9th grade due to his disruptive behavior.

In addition to his habit of wandering on foot, as a teenager JP began to steal the cars of family and friends. Typically, he took cars with keys that had been left in the ignition. He drove in the same direction that the parked car had been facing until the car ran out of fuel. Then he called his parents to pick him up. JP once told Ackerly, "I steer by the compass and never make turns (Ackerly, 1964)." A salient example of JP's inability to sustain relationships occurred in 10th grade when he took a young woman to dinner in Kentucky. While she was in the restroom, he took her purse and went to Alabama.

When JP's problematic behaviors recurred, he was transferred from the public school to a Catholic school. Although he liked the rigid discipline of the parochial school, he was frequently truant. Next, he was transferred to an out-of-state military school. After two months in the military school, JP stole a teacher's car. He was sentenced to two years in a juvenile detention facility ("reform school") (Ackerly, 1964). Following his release and return to Louisville, JP continued his wandering behavior, hitchhiking hundreds of miles and sometimes stealing cars from strangers. His parents would pay for damages and tried to keep him from being jailed (Ackerly, 1964). He never completed tenth grade.

3.1.2. *Young adulthood*

In January 1932, at age nineteen, the police pressed charges against JP for grand larceny when he broke into his family's garage and took the family car after having been denied permission to use it. At a hearing, the judge told the patient that he "would be sent to the workhouse" if any further arrests occurred ("Youth Admonished," 1932).

In August 1933, JP stole the automobile of a neighbor. He abandoned the car in Indiana when the fuel ran out and was arrested again. This time he was jailed and scheduled to appear before a grand jury two months later ("Held to Grand Jury," 1933). In an attempt to prevent a jail term, an attorney who was working with the patient's parents brought JP to Spafford Ackerly at the University of Louisville for a psychiatric evaluation in October 1933 (Ackerly, 1964). JP was twenty years old.

Ackerly felt that JP's presentation merited neurological evaluation. He admitted JP to the psychopathic ward at Louisville General Hospital where JP was observed to be overpolite, boastful, and excessively talkative. He had no anxiety, hallucinations, delusions, bizarre ideas, or conversion symptoms. According to Ackerly and Benton, JP had an excellent sense of right and wrong in the abstract. He was quite impulsive and had an erratic schedule of eating and sleeping. He lacked a sense of a past or a future, could not plan ahead, and had no lasting resentment toward anyone.

Ackerly described JP's memory as "capricious" for both immediate and remote material, with the exception of excellent memory for things related in any way to automobiles. JP confabulated easily, spinning grandiose tales to fit any topic that came up (Ackerly, 1964; Ackerly & Benton, 1948). He insisted that he had achieved great accomplishments in whatever was being discussed, despite the complete irrationality of his claims; his childhood boastfulness persisted into adulthood.

Ackerly and Benton reported that JP's neurological examinations had been normal apart from occasional right-sided hyperreflexia. Tests for aphasia were normal. His Stanford-Binet IQ was 97 without intratest scatter. During JP's 1933 hospitalization, a pneumoencephalogram revealed deformity of the anterior horn of the left lateral ventricle, slight left to right displacement of the third and lateral ventricles, and an air-filled cavity in the right frontal lobe. The pneumoencephalogram was read as consistent with either a degenerative process or an abscess. An exploratory craniotomy was then carried out by neurosurgeon, Glen Spurling. The postoperative diagnoses were: absence of the right prefrontal lobe and cystic degeneration of the left frontal lobe; chronic arachnoiditis; and anterior fossa adhesions (Ackerly, 1964; Ackerly & Benton, 1948). In a 1977 video-taped conversation with Ackerly, JP indicated that he understood from his conversation with the neurosurgeon forty-four years earlier that he had had an "abscess on the frontal lobe of the brain." Ackerly agreed with him (video recording of interview, January 1977).

When Ackerly reported his findings to the court, the charges against JP were dropped and JP returned home to his parents. JP's problematic behaviors continued unabated. He pawned his mother's rings, stole his uncle's car, and drove to Chicago. After threatening his father in a drunken state, JP was committed to Central State Hospital. However, he escaped and made his way to Colorado. Upon his return to Louisville, JP was readmitted to the state hospital. He escaped again and resumed his wandering behavior.

Despite JP's behavioral shortcomings he made a positive first impression. At age twenty-two in December 1935, a social worker accompanied him by train to Yale University for a comprehensive assessment, including an evaluation by neuropsychologist Catherine Miles. The social worker observed him on the train chatting with a young music teacher. When the snack vendor made his rounds, JP asked the young woman if she'd like to have a chocolate bar. He

ordered two candy bars, and then asked the young woman to pay for her own candy bar since he had only enough money for one. The social worker documented the grandiose, "tall tales" JP told other travelers, observing that JP confabulated stories in response to each topic of conversation (Ackerly, 1964).

While at Yale JP wrote letters to his parents in Louisville (Appendix 1). Two examples, written three weeks apart, were contained in Ackerly's files. JP's cursive handwriting was of uniform size and aligned to the left margins. Syntax, grammar, and punctuation were normal. Vocabulary was approximately consistent with early high school level. There were no spelling errors. The letters reflect JP's affection for his family members and his desire to be in touch with aunts, uncles, and cousins. They offer evidence of his affability as when he signs one letter, "your loving bad boy," and "Tell Stoddard that I said for him to stick by his Dad and obey him explicitly or he'll be just like me." The letters also provide more examples of his ability to initiate relationships. JP wrote of having gone to a movie theater with a young woman who worked at Yale. This is reminiscent of a similar event that occurred in 1936, when JP went to Chicago for a neuropsychological evaluation by Ward Halstead and a young woman volunteer at the hospital "became involved" with him (Ackerly, 1964). JP's letters also reflect his simplicity of thought. For example, he described several acquaintances as being friends. The letters also provide documentation for his "Chesterfieldian" attitude: "Well, now to answer your letter to perfection." And "I have met Dr. Kahn and have nothing but the highest regard for him. He is a thorough gentleman, and a wonderful man physically, mentally, and scientifically."

In subsequent years, JP was successful at finding several jobs. These seldom lasted more than a few weeks to a few months. He worked as a gas station attendant, night watchman, factory worker, salesman, bus driver, interstate truck driver, hospital orderly, and ambulance driver. As Ackerly said in his 1964 paper, JP was at his happiest around cars and hospitals. His superficial charm initially won him jobs and pleased his customers or bosses. However, JP would become irritable when asked to attend to two things at once. This difficulty with divided attention along with erratic performance often resulted in him being fired. JP served in the army for 11 months until he was discharged for convulsions, having "run out of pills (Ackerly, 1964)." This is the only mention of convulsions in the extant record, other than the episode of seizures JP experienced at age 4.

JP was arrested twelve times between 1932 and 1944, from ages nineteen to thirty-one, for disorderly conduct, car theft, and drunkenness. None of these incidents involved violence. Ackerly would provide information to the court on JP's behalf, and charges would subsequently be dropped (personal communication, Carita Warner). JP's father tried to prevent further car thefts by buying JP a car, however this was unsuccessful (Ackerly & Benton, 1948). Despite occasionally drinking as a young adult, there is no evidence in the published record of chronic alcoholism.

Several informants, including Ackerly's daughters, grandson (Dr. William S. Smock), and a former student, commented on the obviously warm relationship between Ackerly and JP. JP occasionally visited the Ackerly home, for instance, to show a new car to Ackerly (personal communication, Betsy Wurtman).

One of Ackerly's daughters (personal communication, Carita Warner) recalled a time in 1943 when she was thirteen years old, and JP was thirty. She had just finished a typing lesson at her father's office when she encountered JP on the stairs, and he proposed marriage to her. Ackerly's publications make it clear that, despite JP's severe social deficits, he regarded JP as a sympathetic character. Ackerly wrote, "the world at hand is indeed too much for him [JP]." He referred to JP as "defenseless, vulnerable and helpless," "a veritable stranger in this world with no other world to flee to for comfort (Ackerly, 1964)."

3.1.3. *Middle age*

When JP was thirty-seven years old, Ackerly described his patient's behavior as follows:

> "His mood is uniform; his affable manner, vocabulary, and stereotyped expressions are the same today as 18 years ago-all are monotonously unchanged. He never holds grudges nor speaks ill of anyone, never picks a fight, never tricks anyone. He is drawn neither to underworld characters nor to churchmen. One is struck with the childish simplicity and superficiality of his petty lying and stealing and sex experiences which are unpremeditated … Of all patients encountered he is by far the most stimulus bound. What he does at the moment absorbs his complete attention, and he shows irritation when suddenly asked to shift … His philosophizing is limited to familiar stock phrases such as "a stitch in time," "the early bird catches the worm," etc. … No one can know him intimately without developing a profound sense of sympathy and a curious feeling of warmth and admiration for his child-like simplicity and guileless responsiveness (Ackerly, 1950)."

When JP was forty-six, his father died. From this point forward his wandering behavior was limited to the city of Louisville. Every day JP drank at least 12 cups of coffee and smoked two packs of cigarettes. His appetite waxed and waned, but his weight remained constant (Ackerly, 1964). JP was easily startled by the noise of children in the street and his memory had begun to decrease. In July 1962, when JP was forty-nine, his mother wrote the following in a letter to Dr. Spurling:

> "I am writing to ask you or some member of your staff to do another x-ray on my son's head. Dr. Ackerly calls it an air study or pneumoencephalogram. His recent memory has been failing seriously of late until today he forgets practically everything."

At that time, Ackerly noted that JP would forget having met someone five minutes earlier, yet he was able to recall in detail anything related to his automobile travels for up to twenty-four hours afterwards.

3.1.4. *Later life*

JP lived with his mother who cared for him until the two entered a nursing home together after losing their home in a

1974 tornado. Psychological testing performed in December 1975 when JP was age sixty-two revealed that much of his personality was unchanged from the earlier descriptions in Ackerly's papers. JP continued to be outgoing and talkative. He tended toward grandiose confabulation, saying for example that he had attended Yale and that he and his mother owned the nursing home in which they lived. He accepted the assessment tasks he was given without question and complied readily in doing them. He was polite and gentlemanly, occasionally drawing attention to his own courteousness (e.g. "a man is supposed to get up for a lady").

The psychologist noted that JP demonstrated good table manners when his lunch arrived. In a proud way that befitted a child, he credited this to the influence of his mother. JP was tearful several times when recalling associations with his mother. His recent memory was impaired. For instance, he asked the examiner repeatedly if she minded his smoking and whether she'd like a cigarette, even though she had told him several times that she preferred he not smoke. Overall, the examiner described him as submissive and dependent. She stated, "JP seemed vaguely aware of some personal inadequacy," but tended to portray himself as "a person of adequacy and importance (Report of Psychological Consultation, 1975)."

In January 1977, at age sixty-four, JP was admitted from his nursing home to the University of Louisville inpatient psychiatry service. In the two to three weeks preceding admission, JP began to be aggressive toward staff. This represented a change in his behavior. He also had been incontinent of urine since falling and fracturing his elbow and femur in April 1976.

Neurological examination by Daniel Tucker revealed normal cranial nerves, cogwheel rigidity in the upper extremities, and increased tone and ataxia in the lower extremities. JP's strength was symmetrically intact. Deep tendon reflexes were 2+ bilaterally in the upper extremities. Patellar reflexes were 3+ bilaterally. Achilles reflexes were 1+ bilaterally. Plantar responses were bilaterally extensor. Palmomental and snout reflexes, gegenhalten rigidity and Myerson's sign were all present. His gait was hesitant, shuffling, and short-stepped. Mental status examination revealed a man who appeared indifferent to his situation and flippant in his responses. JP had little spontaneous verbal output. His responses were slow and often tangential. His affect was bland, but he was able to laugh and use sarcasm. Although oriented to person, place, and season, he was off by decades when asked the year. He could not recall any of three words spontaneously, and his remote memory was vague and inexact (Admission Evaluation Note, 1977).

The authors reviewed a videotape of Spafford Ackerly interviewing JP during this 1977 hospitalization. With prompting, JP affectionately recalled details about the various people involved in his medical care in the 1930's and acknowledged that he had stolen many cars. He fondly recalled Ackerly helping him avoid jail for car theft. He and Ackerly were genuinely friendly during their conversation, consistent with the observations of multiple informants.

JP did not spontaneously recall information in response to questions. He was able to fill in details relevant to each question only when prompted. With encouragement, he recalled a song and sang it with reasonable fidelity to the tune, a song he'd recorded as a birthday gift for Ackerly's wife decades earlier (video recording of Ackerly interviewing JP, January 1977). With the many calls to Ackerly whenever JP was in trouble, and JP's occasional visits to the Ackerly home, JP knew Ackerly's family members and was well-known by the Ackerly family.

Cranial CT scanning on January 31, 1977 revealed enlarged ventricles and a left frontal porencephalic cyst. The cyst communicated with the frontal horn of the left lateral ventricle. A large area of right frontal porencephaly communicated with the right lateral ventricle. There was minimal right frontal cortical mantel, and no subdural hematomas were in evidence (Fig. 1). The third and lateral ventricular enlargement noted on the CT was significantly greater than what was seen on the three-year post-operative pneumoencephalogram from 1936. The CT showed evidence of sulcal effacement indicating recent development or expansion of the hydrocephalus. A diagnosis of normal pressure hydrocephalus was made, and a right ventriculoperitoneal shunt was inserted, resulting in some improvement in JP's gait.

Three and a half months after the shunt was inserted, JP died. On May 24, 1977, the day before JP's funeral, his mother sent a note to Ackerly asking that her son's brain be removed and sent to the medical school for study prior to his burial (letter from JP's mother, Ackerly archives). A small team of

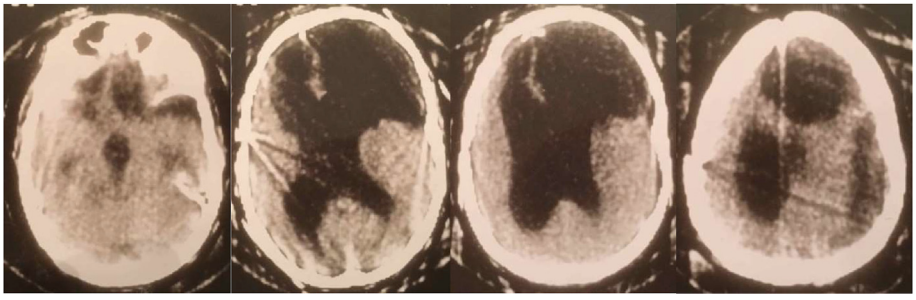

Fig. 1 – Cranial computed tomographic (CT) scan January 31, 1977 (age 64). Note: the right hemisphere is on the right side in these images.

people went to the funeral home to remove the brain and brought it to the University of Louisville pathology laboratory (personal communication, John Rice). JP's body had already been embalmed. The authors discovered that the abstract of JP's brain cutting was missing from the neuropathology case ledger for 1977. No report was ever located.

3.2. *Review of neurodiagnostic testing over JP's lifetime*

On intake at the Louisville Child Guidance clinic in 1926 when JP was thirteen years old, his Stanford-Binet IQ was 92 again without intratest scatter. "His planning ability and capacity to modify behavior by experience" were described as "not equal to that of the average seven-year-old child (Ackerly, 1964)." When given "puzzle-type tests," he perseverated in his approach, unable to inhibit immediate responses in favor of those based on analysis and judgment. At age twenty, his planning ability and foresight were deficient on the Porteus Maze test. However, his performance was superior on the Minnesota Assembly Test, a measure of mechanical aptitude.

During the time that Ackerly cared for JP, he referred him for psychological testing to several prominent psychologists. These included Arthur Benton, W. Horsley Gantt, Ward Halstead, Catherine Cox Miles, Brenda Milner, Ralph Reitan, and James Whitman. Reports of the actual examinations were not found despite an exhaustive search of Ackerly's personal papers. From Ackerly's published reports, JP's IQ was consistently estimated to be in the 90–105 range.

In three repetitions of the Wisconsin Card Sorting Task given at different times by Brenda Milner, JP first sorted quickly without regard to form, color, or number, and then on two subsequent administrations he perseverated on form through the entire 128 card deck. In 1936, Ward Halstead concluded that JP was seriously impaired, based on his testing battery. Halstead commented on how stereotyped JP's behavior was in all interactions.

A 1938 examination by W. Horsley Gantt when JP was twenty-five found impairment in new learning ability. In 1960, Ralph Reitan estimated JP's IQ at 105 and noted that JP did poorly on a category test. He also had mild bilateral impairment in tapping speed.

The following year psychologist James R. Whitman noted that JP, now forty-nine or fifty years old, had problems recalling what they'd done two days earlier. Whitman noted that the full-scale score was lowered by subtests requiring new learning, copying block designs, and assembling parts into a meaningful pattern. Despite reports that JP had a good overall memory in childhood, there were at least some deficits in new learning by age twenty-five (Ackerly, 1964).

Ackerly and Benton summarized JP's multiple psychological evaluations from ages eleven to thirty as showing: no intellectual deficit despite his massive frontal damage, a "nonorganic" performance on mental test batteries, but defective performance on tasks requiring planning and keeping a remote goal in mind, such as the "ball and field test" of the Stanford Binet battery (Ackerly & Benton, 1948).

JP lacked social anxiety in many life situations. In 1962, at age fifty, his stress hormone response was studied at Walter Reed by John Mason. For this study—with methodology that would raise questions today—JP was locked in a psychiatric unit overnight and exposed to a stressful interview. Urinary 17-hydroxy corticosteroid levels measured before, during, and after the interview were twice that of controls (Ackerly, 1964). This demonstrated that JP did in fact exhibit a physiological marker of stress despite not being known to have ever manifested external signs of anxiety.

Electroencephalograms (EEG) at ages twenty-four and twenty-eight were read as normal, but Ackerly felt the first recording was substandard. The former study had some bursts of slowing that were not read as abnormal at the time as they were of low amplitude. The record of the latter study could not be located (Ackerly & Benton, 1948). In the context of being treated with phenytoin for seizures, an EEG at age forty-nine showed "general dysrhythmia and an irritative left frontal temporal focus (Ackerly, 1964)." Other than the mention of JP having "run out of pills" while he was in the military, this is the only documentation of JP having been medicated for seizures. When considered together with JP's seizure semiology of left-sided onset at age four, this indicates that JP likely had bilateral epileptic foci. Unfortunately no other data concerning JP's seizures was located.

The December 1975 psychological evaluation of JP at age sixty-three included a WAIS Full Scale IQ of 83, with a Verbal IQ of 90 and a Performance IQ of 76. His performance was very slow on a Bender Gestalt task of visual motor dexterity. He drew primitively and had problems in perceptual organization. JP blamed his inability to draw on his vision. On the Wide Range Achievement Test (WRAT) reading subtest, he achieved a grade equivalent level of 13. The spelling subtest was abandoned at the fifth-grade level due to response slowness and distractibility. The examiner estimated that JP's spelling was consistent with his reading level and commented that he was deficient at planning and keeping long range goals in mind.

In a subsequent evaluation at age sixty-four in January 1977, JP's verbal IQ was measured at 85, with performance IQ at 63, for a full-scale IQ of 74, demonstrating a decrement in intellectual ability in his final years of life.

A timeline of known events in JP's life is provided in Table 1.

3.3. *Neuropathologic findings and interpretation*

The authors were unsuccessful in locating the brain of JP. However, we were able to find a video recording and photographs of the brain cutting that was done by neuropathologist Ryland Byrd at the University of Louisville. The following description is based entirely on a review (by TWS) of all available photographs and the video recording of the brain cutting.

3.3.1. *Skull and dura mater*

Externally, the calvarium shows evidence of an old left frontoparietal craniotomy incision and a small right parietal burr hole through which passes a ventricular catheter. With removal of the calvarium, bilateral subdural hematomas are seen. These are subacute or chronic as evidenced by the presence of yellow-brown membranes adherent to the inner

Table 1 – Timeline of events in JP's life.

Date	Age	Information
December 29, 1912		JP is born at home after 22-h labor, weighing 11.5 pounds, instrumentation required, maternal lacerations
1913	9 days	6.5-pound weight loss since birth, jaundiced
1914	1	Walking and talking normally
1915	2 1/2	Wandering behavior begins
1916	3	Ran away from home & returned by police several times
1917	4	Struck head jumping off bed; 1 h later developed confusion, loss of consciousness, left-sided seizures, resolving in 4 h
1919	6	Defecated in a paint pot in school
1920	7	Defecated in a child's glove and overshoe; masturbated in class; transferred to another school
1924	11	Tested by school system research bureau due to adjustment problems; Stanford Binet IQ 90
1925	12	JP diagnosed as having psychopathic personality
1926	13	Transferred to day school for "defective children;" evaluation at Louisville Mental Hygiene Clinic
1927–1930	14–17	Did well at small private school, transferred to public high school skipping grade 9
1927–1930	14–17	Returned to 9th grade for behavioral problems
1927–1930	14–17	Transferred to Catholic school for better discipline
1927–1930	14–17	Transferred to out of state military school due to truancy at Catholic school
1927–1930	14–17	Sentenced to 2 years in reform school for stealing teacher's car 2 months after starting military school
	Unknown	Following release JP resumes "wandering," hitchhiking thousands of miles, stealing cars, father paying damages to prevent legal charges being filed
1932	19	Police press charges for grand larceny after JP breaks into father's garage and takes car
1933	20	Arrested for grand larceny after stealing a neighbor's car
1933	20	Attorney brings JP to see Dr. Ackerly
1933	20	Admitted to psychopathic ward for evaluation; Pneumoencephalogram shows large left frontal defect; Exploratory craniotomy by Glen Spurling: cystic degeneration of left frontal lobe, absence of right prefrontal lobe
1933	20	Pawned mother's rings, took uncle's car, drove to Chicago
	Unknown	Committed to State Hospital after threatening his father
	Unknown	Escapes from hospital & wanders to Colorado
1935	23	Social worker, Ruth Mellor, takes JP to Yale by train for evaluation by Catherine Miles
1936	23	Neuropsychological evaluation by Catherine Miles at Yale
1936	23	Repeat pneumoencephalogram unchanged
1936	24	Neuropsychological evaluation by Ward Halstead in Chicago
1937?	Unknown	JP serves 11 months in the army
1937	24	EEG read as normal; Ackerly questioned this conclusion
1938	25	Evaluated by Horsley Gantt at Johns Hopkins
1938	25	Escapes with 2 other patients from Central State Hospital in Lakeland, Kentucky (date of admission unknown)
1941	28	EEG read as normal
1932–1944	20–31	JP arrested 12 times
1947	34	Ackerly and Benton present "A Case of Bilateral Frontal Lobe Defect" at Association of Nervous and Mental Disease meeting on the Frontal Lobes (Ackerly & Benton, 1948)
1950	37	Ackerly publishes "Prefrontal Lobes and Social Development" including data about JP (Ackerly, 1950)
1959	46	JP's father dies
1960	47	Neuropsychological evaluation by Ralph Reitan
1961	48	Psychological evaluation by James Whitman
1962	49	JP's mother writes to Glen Spurling requesting a repeat pneumoencephalogram to investigate JP's recently deteriorating memory
1962	49	EEG shows generalized dysrhythmia and irritative left fronto-temporal focus
1962	49	Neuroendocrine evaluation by John Mason at Walter Reed
1964	51	Ackerly publishes "A case of paranatal bilateral frontal lobe defect observed for thirty years"
1974	61	JP moves with his mother to nursing home after their house is destroyed in a tornado
1975	62	JP falls in nursing home
1975	62	Psychological evaluation by Edith Bloch, MA in Louisville, KY
1976	64	JP falls and fractures elbow and femur; developing gradually increasing urinary incontinence and gait instability
1977	64	Transferred from nursing home to in-patient psychiatry
1977	64	Video recording of Ackerly interviewing JP on psychiatric unit
1977	64	Brief psychological evaluation by Regina O'Daniel, MA at Louisville General Hospital
1977	64	Ventriculoperitoneal shunt placed for presumed normal pressure hydrocephalus
May 24, 1977	64	Death of JP 3 1/2 months after shunt placed
1977		Letter from JP's mother giving permission for brain removal; Brain cutting by Ryland Byrd

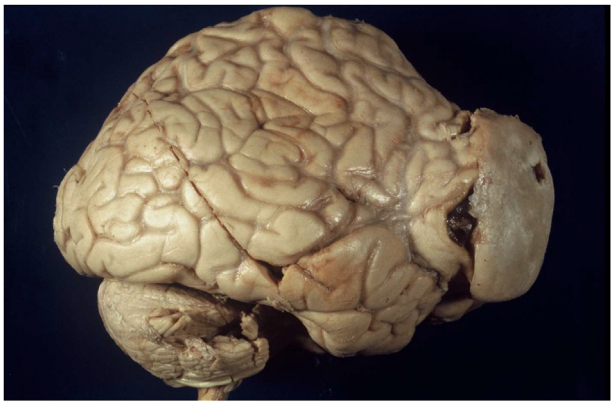

Fig. 2 – Lateral view of the right cerebral hemisphere. The right frontal lobe anterior to the central sulcus is deformed, most likely due to compression from a subdural hematoma. Some hemosiderin-staining is also present in this region. The prominent extension of the right frontal pole contains old blood clot and is densely adherent to the dura.

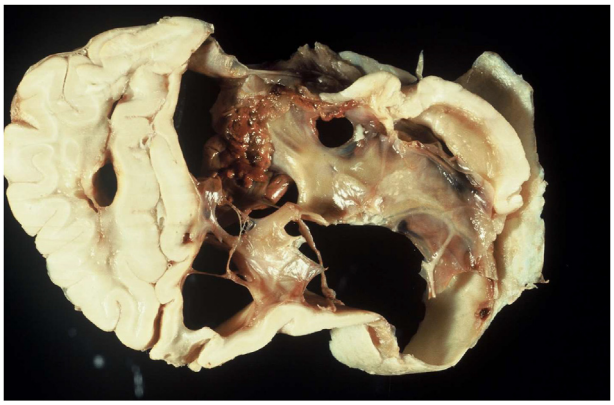

Fig. 3 – Coronal section through anterior cerebrum showing extensive, partly fenestrated cavitation of the entire right frontal lobe with some old hemorrhage present along the medial and superior aspects. The right frontal cortex is markedly attenuated or absent, and no white matter is evident. The left frontal cortex is better preserved but the white matter is markedly shrunken and gliotic. Hemosiderin staining is present in the left frontal horn of the ventricle.

surface of the dura. Embedded within the membranes are several more recent-appearing hemorrhages.

Both cerebral hemispheres are deformed and compressed laterally, more so on the left. We presume this is secondary to the prior subdural hemorrhages. After brain removal, a small amount of subdural hemorrhage is noted at the base of the left middle cranial fossa. The basal calvarium is otherwise unremarkable. The presence of any healed skull fractures or other deformations cannot be ascertained since the dura had not been removed from the base.

3.3.2. *External brain examination*

The anterior frontal dura is densely adherent to the underlying brain. Upon incising the dura, there is extensive destruction, distortion and loss of brain tissue involving both frontal lobes, more so on the right, extending from the frontal poles and superior lateral frontal lobes posteriorly for an estimated 2–3 cm (Fig. 2). Slight hemosiderin-staining is present in the leptomeninges overlying the bilateral posterior frontal and lateral temporal lobes. The surface anatomy in these regions is unremarkable, as is the remainder of the cerebrum, cerebellum, and brainstem. No brain swelling or true herniations are present. There is deep grooving of the bilateral unci, possibly the result of some downward displacement of the brain from prior subdural hemorrhages. The Circle of Willis vasculature is unremarkable. The optic nerves, chiasm, and other visualized cranial nerves appear normal. The base of the hypothalamus is difficult to visualize. The mammillary bodies cannot be seen.

3.3.3. *Coronal sections of brain*

The coronal sections confirm that the most severe damage involves the right anterior frontal lobe (Fig. 3). In this area there is nearly complete tissue loss with formation of a large partially fenestrated cavity. The cavity walls are formed by both dura and a thin rind of atrophic cerebral cortex. The

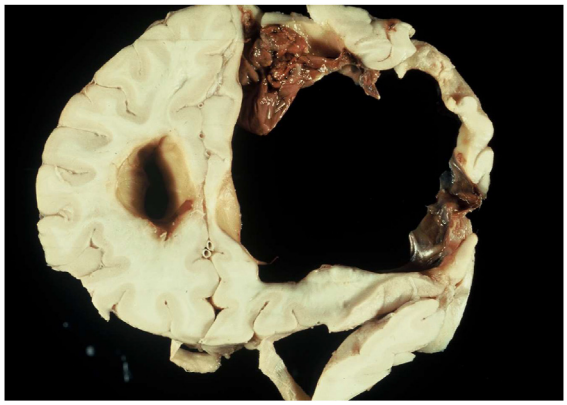

Fig. 4 – Coronal section through mid-frontal lobes (level posterior to Fig. 3) showing marked cavitation of the right frontal lobe with old hemorrhage along the superior-medial and lateral aspects of the cavity. Markedly attenuated or absent cortex is present, along with complete absence of the white matter. The left frontal lobe shows normal-appearing cortex and relatively intact but moderately attenuated white matter. The left lateral ventricle is enlarged and shows some hemosiderin-staining. A connection or boundary between the right cavitary lesion and lateral ventricle is not evident in this section.

internal aspect of the cavity shows hemosiderin-staining and a small old, brown-colored hematoma at its superior medial margin (Fig. 4).

More posterior sections indicate that the cavitary lesion is continuous with the frontal horn of the right lateral ventricle which is considerably enlarged (Fig. 5). The frontal white

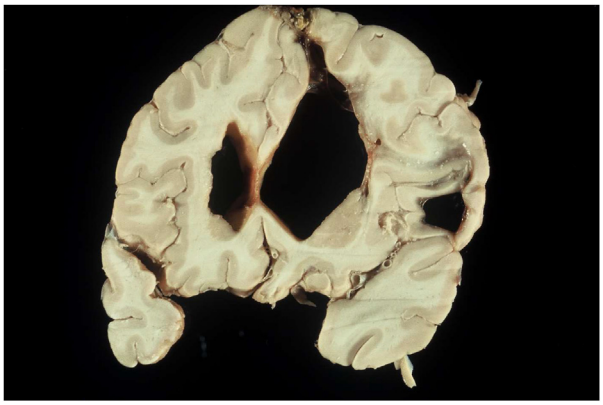

Fig. 5 – Coronal section of cerebrum (posterior to Fig. 4) showing the probable site of communication between the right frontal cystic cavity (superior) and a markedly enlarged frontal horn of the right lateral ventricle. The frontal cortex at this level is better preserved, however the white matter is markedly attenuated and gliotic. The left frontal cortex shows normal thickness, but the white matter is reduced in volume and the left frontal horn is enlarged. Both ventricles show some surface hemosiderin-staining. The rostral corpus callosum (genu) is atrophic.

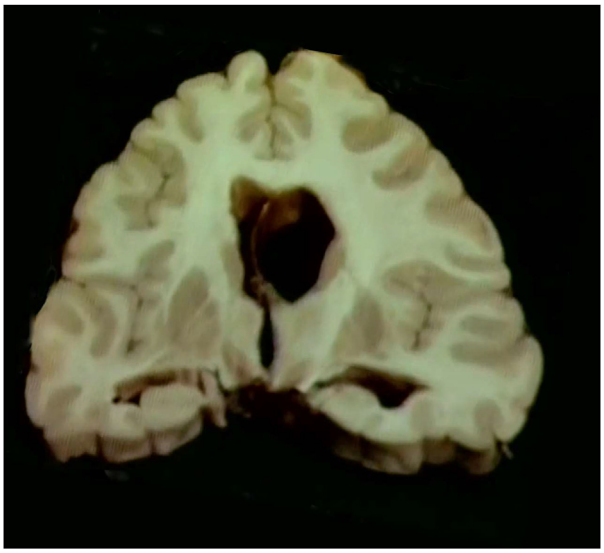

Fig. 6 – Coronal section at mid-cerebral level (posterior to right frontal cystic lesion) showing ventricular enlargement (greater on the right) with hemosiderin-staining of lateral ventricles and fenestrated septum pellucidum. The fornices are difficult to identify but probably atrophic. There is moderate volume loss of the right hippocampus. The cortical surfaces bilaterally are slightly flattened, but the cortex is otherwise well-preserved. The cerebral white matter, basal ganglia and thalami likewise appear normal (accounting for slight asymmetry of the coronal section.) (Note: image obtained directly from video of brain cutting session).

matter at this level shows considerable volume loss and gliosis, however the cortical ribbon is near normal in thickness.

The left frontal lobe shows less severe damage, with a smaller slit-like hemosiderin-stained cavity occupying most of the white matter. Posteriorly, it has slightly less gliosis and ventricular enlargement.

The rostral corpus callosum is thin and gliotic. The septum pellucidum is relatively intact but appears stretched and thin. It shows extensive hemosiderin staining. The fornices are difficult to visualize due to their marked atrophy.

The video images of the coronal sections show diffuse ventricular enlargement with hemosiderin discoloration of the ependymal surfaces. The basal ganglia are of normal volume and configuration. The mid- and caudal thalami are normal in volume. The rostral thalami, including the anterior and dorsomedial nuclei, appear slightly reduced in size.

The outlines of the mammillary bodies are difficult to discern but are likely severely atrophic. There is no indication that they were torn away during removal of the brain. The medial temporal lobe structures including amygdala and hippocampi are difficult to evaluate on the video images. The temporal horns are clearly enlarged, possibly greater on the right. The bilateral amygdalae appear relatively normal in size and shape.

Both hippocampi are slightly distorted or compressed by the ventricular enlargement. This enlargement complicates accurate assessment of hippocampal volume. A discrepancy in hippocampal size is noted posteriorly, the right appearing smaller (Fig. 6). This suggests that the right hippocampus may have some tissue loss. In the posterior temporal, parietal and occipital lobes, white matter volume and cortical thickness appear relatively normal. Cross sections of the cerebellum and brainstem show some enlargement and hemosiderin-staining of the cerebral aqueduct and 4th ventricle, but no other abnormalities. The substantia nigra and locus coeruleus show normal pigmentation.

3.4. *Interpretation of pathologic findings*

3.4.1. *Mechanism of frontal lobe damage*

The rostral frontal lobe damage is almost certainly an acquired lesion and is not of congenital or neurodevelopmental origin. This damage probably occurred during the perinatal or immediate postnatal period. It is most likely the result of external traumatic injury to the head, which was most likely associated with the delivery process. Although it is difficult to reconstruct the precise mechanism of this injury, we postulate that the use of instrumentation during the delivery could have caused severe compression and/or deformation of the skull. In turn this could have resulted in tearing of a dural sinus, which would in turn lead to subdural and/or intracerebral hematomas, and probable hemorrhagic contusions of the anterior frontal lobes.

3.4.2. *Mechanism of ventricular enlargement*

The ventricular enlargement may be a consequence of the perinatal frontal lobe hemorrhagic lesions. The lesions communicated with the lateral ventricles anteriorly, and the presence of hemosiderin pigment along much of the ependymal surface suggests the probability of outflow obstruction occurring either at the brain base or convexities (i.e. communicating hydrocephalus). The ventricular enlargement likely worsened in his last year of life when he developed the characteristic triad of symptoms of normal pressure hydrocephalus subsequent to a fall.

3.4.3. *Mechanism of forniceal atrophy*

There is marked atrophy involving virtually the entire fornix. It is difficult to determine the mechanism of the forniceal injury. A plausible explanation may be that one or several episodes of intraventricular hemorrhage might have taken place at the time of the frontal lobe injury. This hemorrhage would have led to diffuse hemosiderin deposition within the ependyma and anatomic structures abutting the ventricles. The hemorrhage could also have created communicating hydrocephalus.

Both processes, intraventricular hemorrhage and communicating hydrocephalus, could have damaged the columns of the fornix. Ventricular enlargement then likely caused mechanical deformation and stretching of structures attaching to the fornix, such as the septum pellucidum. The hemosiderin deposition could have caused direct toxic damage to the fornices in a manner analogous to CNS superficial siderosis.

4. Discussion

It was in the spirit of Ackerly's 1964 comment that "The last chapter in the history of our patient, JP, will not be written, of course, until later" (Ackerly, 1964) that the authors pursued information that might allow the "last chapter" to be written. Ackerly was present at the brain cutting of JP, along with several coauthors of this paper—Drs. Adams, Rice, Smock, and Tucker. Ackerly arranged for photographs and a video of the procedure. Ackerly also had conversations with one of the coauthors (CBLA) about a follow-up report of the brain cutting. With this in mind, in 1977 Ackerly gave her many of the materials from his files on JP.

After the initial report of the case of JP in 1947, many questions remained unanswered. Was JP's frontal syndrome the result of perinatal damage or his fall at age four? If there was perinatal damage, what was the cause? Was JP's brain damage limited to the frontal lobes? With Ackerly's subsequent 1964 report, a question arose as to the etiology of the gradually progressive anterograde amnesia that developed when JP was forty-nine. To what degree was JP's case representative of other published cases of early life frontal damage? Finally, what was JP's probable cause of death?

4.1. *What was the cause of JP's brain injury?*

Ackerly and Benton reported two potential causes of JP's brain damage although they favored the first: perinatal injury and a brain injury sustained in a fall at age four. JP's high birthweight would be consistent with maternal gestational diabetes and could explain the need for instrumentation that was employed at birth. It is likely that JP suffered from a major perinatal hemorrhagic event, most likely caused by birth trauma from instrumentation. The damage included bilateral lobar hemorrhages and possible subdural hematomas. A torn venous sinus from distortion of the skull during delivery could explain the bilateral lobar hemorrhages. The breakdown of blood products due to massive brain hemorrhage from venous sinus rupture, as proposed by the authors, then would have caused the early weight loss and jaundice reported on day nine of life. These data are consistent with Ackerly's opinion that JP's pathology was likely perinatal in origin. The absence of any mention of external bruising at birth could mean the information was omitted by or unknown to Dr. Ackerly. However external bruising does not necessarily occur with forceps deliveries, even if traumatic.

Another cause of massive perinatal brain injury could have been an abscess. Brain hemorrhage as a primary presentation of cerebral abscess is exceedingly rare and not supported by the presentation in this case. Unusual infections like invasive fungal abscess or the rupture of a mycotic aneurysm, in theory, could result in hemorrhage, but fever would have occurred, and hemorrhage would not have been the main presenting feature. A primary hemorrhage with secondary abscess formation is also extremely rare and, again, would have included fever. Finally, an abscess as large as the damage shown in JP's brain would have caused profound sepsis and a likelihood of death in early childhood.

JP's unusual behaviors, such as wandering and Chesterfieldian manners, began before his seizure at age 4, providing strong evidence that this was not the causative event. Given JP's rapid and complete return to baseline in less than a day, his left-sided seizures and subsequent period of loss of consciousness at age four appear to have been caused by striking his head and activating a pre-existing seizure focus. Rapid resolution of symptoms would not have been consistent with new hemorrhagic damage.

4.2. *Was JP's brain damage limited to the frontal lobes?*

The coronal sections in figures three through five demonstrate massive cavitary lesions of the right greater than left frontal lobes. The more posterior coronal section in figure six includes the relatively intact posterior frontal and temporal lobes. However, the temporal horn of the right lateral ventricle is somewhat larger than the left, consistent with medial temporal atrophy. This suggestion of hippocampal atrophy and possible bilateral forniceal atrophy might explain why both Gantt and Whitman, examining JP in 1938 and 1961

respectively, found his anterograde learning to be impaired. There is also evidence of extracerebral compression due to subdural hematoma, but the cortex and subcortical white matter are intact. More posteriorly the brain appeared normal. Although it would be difficult to ascertain when the hippocampal atrophy began, it is possible that JP had some medial temporal damage in addition to his massive prefrontal damage. This could have been secondary to his original hemorrhage or due to the effects of gradually increasing hydrocephalus. Bilateral amygdala hypofunction has been associated with diminished capacity for anxiety and fear (Adolphs et al., 1994). The available evidence does not indicate whether JP's right medial temporal atrophy was present from birth or developed secondarily, later in life, but it is at least possible that medial temporal damage contributed to some of JP's social deficits.

4.3. *Why did JP develop progressive anterograde amnesia in middle age?*

The anterograde amnesia that was noted when JP was forty-nine years old stands in contrast to the descriptions by JP's parents and teachers of his good memory in childhood. However, neuropsychologist W. Horsley Gantt observed deficits in new learning as early as 1938 when JP would have been twenty-five years old, perhaps indicating the beginning of an amnestic syndrome that was observed by his mother to have acutely worsened by age forty-nine. There were no known subsequent neurological events and no reports of status epilepticus or cerebral infection that might have caused the anterograde amnesia in middle age. JP was not known to abuse substances. The possibility of a coincident dementing illness cannot be ruled out though no evidence of deterioration in functioning other than in episodic memory was found prior to the incontinence and falling beginning at age sixty-three. The normal pressure hydrocephalus with which he was diagnosed at age sixty-four could also have been gradually accumulating for many years.

4.4. *Was JP representative of other known cases of childhood frontal injury?*

Eslinger, Flaherty-Craig and Benton (Eslinger et al., 2004) and Anderson et al. (Anderson et al., 2009) showed that JP's social adaptation deficits were consistent with those in more recently described cases of early childhood damage confined to the frontal lobes (Anderson et al., 1999, 2006, 2009; Bahia et al., 2013; Eslinger et al., 1992, 1997, 2004; Grattan & Eslinger, 1992; Marlowe, 1992; Mateer & Williams, 1991; Price et al., 1990; Williams & Mateer, 1992). Anderson et al. (2009) studied seven people who had had prefrontal damage between birth and age five to determine whether their intellectual and behavioral profiles were similar to those of JP. Five of the seven were similar to JP in that they had relatively intact intellectual function but impaired social function. Four of the seven had bilateral frontal damage and the other three had unilateral right frontal damage. One patient had extension of damage into the right temporal pole. Individual case histories were not provided but the authors demonstrated a number of similarities between their cases and that of JP. In an earlier 2006 paper (Anderson et al., 2006), Anderson et al. reported four patients with childhood-onset ventromedial frontal damage who had relatively intact intellectual functioning but impaired emotional and social functioning. Detailed behavioral descriptions that would have allowed comparison to JP were not included. It is unclear whether some of these patients were also reported in their 2009 study (Anderson et al., 2009).

Of the cases of early frontal lobe damage with detailed behavioral descriptions reported in the literature, only two, besides JP, involved damage during infancy (GK in the first week of life and ML at 3 months). GK, like JP, had wandering behavior and once stole a car that was parked at a gas station with the keys in the ignition and the gas hose still connected. In contrast to these patients with very early injury, the patients who sustained frontal injuries later in childhood tended to have more social relationships, however problematic. Bilateral frontal damage in childhood, as occurred in both JP and GK, has been associated with more severe cognitive and behavioral deficits than unilateral damage (Eslinger et al., 2004).

None of the patients with childhood frontal lobe damage were reported to have had "Chesterfieldian" politeness, repetitive car theft, or a habit similar to JP's driving solely in one geographic direction. In addition, although JP would become irritable when asked to do two things at once, he lacked a history of aggressive reactivity that characterized the majority of cases of childhood frontal lobe damage.

4.4.1. *Primary deficits in social cognition*

JP's case demonstrates that early life frontal damage can cause permanent impairment in social adaptation and social cognition. Social adaptation can be defined as "adjustment to the demands, restrictions, and mores of society, including the ability to live and work harmoniously with others and to engage in satisfying social interactions and relationships" (American Psychological Association, 2022). Measures of social adjustment seen in the context of psychosocial rehabilitation include ability to sustain interpersonal relationships, maintain oneself in a vocation, and live independently (Harvey et al., 2007).

JP was never able to sustain cooperative interpersonal relationships, keep a job, or live independently. He exhibited an ability to form initial, superficial connections with others. His behaviors were stereotyped and child-like. His thought processes were concrete, especially lacking in foresight or planning. He was never goal oriented. Instead, his behavior was driven by responses to the most recent or salient stimuli he encountered.

Social cognition is defined by Ziaei et al. as the ability to perceive, understand and respond appropriately to the thoughts, feelings, and behaviors of others (Ziaei et al., 2022). JP acted without thinking about how others might be affected. He lacked theory of mind and the capacity for moral reasoning. Deficits in theory of mind have been linked to frontal dysfunction, especially those deficits in the right

hemisphere (Hamilton et al., 2017; Happe et al., 1999; Stuss et al., 2001). The neonatal destruction of JP's right more than left prefrontal cortex likely explains his major deficits in social adaptation and social cognition.

4.4.2. *Moral reasoning*

The impulsive, socially aberrant sexual behaviors of JP's childhood ceased to be prominent in adulthood. But his deficient moral reasoning continued for the rest of his life. The ability to engage in moral reasoning has been associated with the right ventromedial orbitofrontal cortex (Mendez, 2009), an area severely damaged in JP's case. JP was able to articulate and identify what was generally considered right or wrong, perhaps as a consequence of rote learning. However, he was unable to apply this knowledge to his own behavioral choices. JP's limited ability to plan, to foresee consequences of action, or to understand the effects of his choices on others would have undermined any ability to put knowledge of right and wrong into action.

4.4.3. *Perseveration, stimulus salience, and stereotyped behavior*

The perseveration JP demonstrated in multiple neuropsychological evaluations mirrored his perseveration in stealing cars and driving them in the same geographical direction that they were facing until the fuel ran out. His tendency to take cars similar to the ones his father sold and to take unlocked cars with the keys readily visible in the ignition are evidence of JP's tendency toward stimulus bound behaviors driven by the salience of the stimulus. The episode of JP taking his date's purse in tenth grade while she was in the powder room is also consistent with this mechanism. Stimulus bound behavior is typically seen with dorsolateral prefrontal dysfunction. The stereotyped nature of JP's interactions with others is similar to behavior reported in frontotemporal degeneration (Ames, 1998).

4.4.4. *Possible origins of JP's unique traits*

The physical and emotional environment in which JP was raised likely contributed to the origin of some of his more unusual symptoms. His preoccupation with cars can likely be traced to his frequent exposure to the high-end touring cars his father sold at his auto dealership (Chester, 1930). JP confirmed in the 1977 interview with Ackerly that he favored stealing cars like the ones his dad sold.

JP's wandering behavior also was unique and might best be described as arising from a combination of impulse—driven by stimulus salience (Anderson et al., 2009)—and perseveration, in clear consciousness, and without anxiety. JP's wandering began when he was a young child, and his later driving and hitchhiking behaviors could be similarly conceptualized. JP was able to operate an automobile without damaging the vehicle or being apprehended for erratic driving, but, given that he drove in whatever direction the car was facing and stopped when the car ran out of fuel, one may conclude that JP did not have a destination as a goal. Wandering behavior was reported in the case of GK who suffered from frontal damage that was discovered in the first week of life, but no details of the wandering are provided (Price et al., 1990). Wandering behavior with amnesia for the wandering commonly occurs in fugue states and occasionally has been reported as a post-ictal automatism in epilepsy (Mayeux et al., 1979). Wandering is also common in Alzheimer disease and frontotemporal dementia and some authors have considered it a form of nonverbal perseveration (Ryan et al., 1995); however, in

Table 2 – Summary of JP's cognitive deficits and behavioral characteristics.

Impaired cognitive functions
Limited sense of past or future ("lived in the present")
Absence of planning or foresight
Inability to consider consequences of behavior
Limited ability to learn from experience
Impulsivity
Lack of motivation
Failure to complete tasks (distractibility)
Deficient divided attention (became irritable when asked to attend to 2 tasks at once)
Concrete thought
Perseveration
Stimulus bound behavior
Placidity, Equanimity, Docility
Lack of social anxiety or fear
Impaired emotional expression with limited display of enjoyment or enthusiasm
Impaired social cognition
Inability to consider effects of his behavior on others
Inability to take others' perspectives (theory of mind)
Lack of insight
Impaired moral reasoning as to his own behaviors (preserved ability to define moral behaviors in general)
Impaired social adaptation
Interpersonal relating characterized by:
Interpersonal communication deficits
Overreliance on aphorisms
Stereotyped "Chesterfieldian" (superficial and excessively polite) manners
Boastful/grandiose (confabulated personal achievements)
Garrulousness
Inability to form intimate attachments with others
Limited ability to form cooperative relationships (including in school)
Inappropriate sexual behavior in childhood
Poorly developed sense of responsibility
Vocational experiences characterized by:
Inability to sustain job performance
Repeatedly fired from jobs
Only short stints at jobs he sought as bus driver, truck driver, ambulance driver, gas station attendant, hospital orderly
Daily functioning characterized by:
Tendency to aimlessly wander great distances
Living with mother until late middle age, then living in same nursing home as mother
Erratic eating and sleeping
Absence of adolescent rebellion against authority or desire for independence
Difficulty managing money

contrast to JP, individuals with these disorders are unable to describe the details of their wandering.

Although a purely psychological hypothesis cannot be proven, one clue as to the possible origin of JP's "Chesterfieldian" manners may be found in an observation made by Ackerly and Benton about JP's father. "He was impatient with his young son, demanding perfect behavior and adult standards of conduct from an early age (Ackerly & Benton, 1948)." JP's "Chesterfieldian" manners were both stereotyped and reflective of JP's inability to vary his behavior based on social context. The same stereotyped manners were still present in the aforementioned psychological evaluation at age sixty-three. In summary, JP's unique behaviors likely arose from a combination of influences, including his innate temperament, intelligence, rearing as a child, his psychosocial environment, and his severe frontal damage.

A summary of JP's cognitive and behavioral deficits is presented in Table 2.

4.4.5. *Strengths*

Although JP's neuropsychological and neurobehavioral deficits are well described, he also had several relative strengths. As Ackerly said, "The wonder is, however, not the state of disability exhibited by these patients, but rather the semblance of normality at any one moment (Ackerly, 1964)." JP was able to initiate relationships and could seem normal in a brief meeting. This was apparent in the social worker chaperone's description of his behavior on the train to Yale. The 1975 psychology report captured some of JP's strengths, describing him as a "pleasant and friendly man," "trusting and sensitive" (Report of Psychological Consultation, 1975). Also, the tone in Ackerly's publications indicate a fondness for his famous patient. The warmth of their relationship was captured in the 1977 video-recorded conversation.

The letters written to family from Yale at age twenty-three are consistent with reasonably good verbal fluency and expressive language despite his perinatal left frontal damage. The quality of the writing in JP's letters suggests a degree of neuroplasticity. They reflect a close relationship with his parents, attachment to other relatives, affability, and a simple sense of humor. The letters also demonstrate JP's fondness for Ackerly. In the letter, which can be found in Appendix 1, he asks his mother to "give my regards to Dr. Ackerly and tell him that I am anxious to see him."

Interviews with Ackerly's daughters verified that their father was frequently called to intercede when JP was in trouble and that Ackerly felt warmly protective of JP. They also reported that, when JP's father bought him a car, JP drove to the Ackerly home to show it to his doctor. JP's ability to make a good first impression allowed him to obtain jobs relatively easily. The happiness JP showed when working in hospitals or driving cars likely reflected the warmth of his relationship with Ackerly and the closeness he felt to his father when around cars.

4.5. *How did JP's behavior differ from other serious childhood behavioral disorders?*

JP's remarkable behavior included features that could be confused with conduct disorder, antisocial personality, or autism spectrum disorder. The DSM-5-TR diagnosis of conduct disorder requires three of fifteen criteria present in the year prior to diagnosis with at least one present in the six months prior to diagnosis (American Psychiatric Association, 2022). These criteria fall into four different categories: aggression to people and animals; property destruction; deceitfulness or theft; and serious rule violations. The first two criterion groups require the actions to be deliberate and cruel. JP's compulsive car theft could possibly be construed as breaking into cars and theft "without confrontation of the victim" as specified in the deceitfulness or theft category, as he only took cars that were unlocked with keys visible and he was not motivated by personal gain. Although truancy is one of the criteria in the serious rule violation category, JP would not have met the required three criteria for conduct disorder. Antisocial personality disorder is preceded by conduct disorder prior to age fifteen. In addition, personality disorders are not diagnosed if the behaviors are attributable to another medical condition, and JP clearly suffered from a serious brain injury. In contrast to people with antisocial personality disorder, JP did not have a pervasive pattern of disregard for and violation of the rights of others and was not deceitful or manipulative. He was unmotivated by financial gain, did not seek to con or manipulate others, and was not arrogant. His stealing the purse from his date in tenth grade, like his stealing the cars of family and neighbors, appears to have been driven by the immediacy or salience of the stimuli, and not by criminal intent. Despite having behaviors that were superficially similar to antisocial personality, he was polite and considerate in the moment and did not seek advantages over others. He was also placid, docile, and lacked social anxiety. He had neither a sense of past nor of future. He did not appear to learn from experience. None of these features are common in antisocial personality disorder, despite JP's having been diagnosed with "psychopathic personality" at age twelve (Ackerly, 1950). Ward Halstead commented to Dr. Ackerly in 1950 that JP was more asocial than antisocial (Ackerly, 1950).

JP's repetitive behaviors and stereotyped interpersonal relations and his apparent lack of theory of mind could be seen as overlapping with autism spectrum disorder (ASD). The similarity is only superficial, however. The core features of ASD are persistent deficits in social interactions and restrictive repetitive behavior patterns. JP's repetitive behaviors were not restrictive as in ASD. JP could be charming and endearing, and capable of forming superficial relationships initially. However, apart from his asymmetric relationships with Ackerly and care-taking family members, these relationships would be unsustainable due, among other things, to his inability to consider "what effects his past and present thoughts, feelings and behavior might have on the future of that relationship (Ackerly, 1964)."

4.6. *What caused JP's death?*

JP's death certificate lists pneumonia as the proximate cause of death. However, the post-mortem brain examination revealed evidence of relatively recent, large, bilateral subdural hematomas. Bilateral subdural hematomas of this magnitude would have caused severe neurological compromise and would have been seen on his January 1977 cranial CT scan had

they been present. Instead, placement of the ventriculoperitoneal shunt three and a half months prior to JP's death likely resulted in drainage of large amounts of cystic fluid, rapid reduction in ventricular volume, and consequent collapse of the cerebral hemispheres.

4.7. *Limitations of this study*

In reconstructing the events of JP's life without being able to examine the patient, the authors relied on the published observations of Drs. Ackerly and Benton and previously unreported data including: video footage, photographs, newspaper articles, interviews of witnesses, and data from the Ackerly archives. There is much medical information that could not be located but would have been useful to review. We were unable to find complete information about JP's seizure disorder. Neither the actual reports nor the raw data from JP's many neuropsychological evaluations reported by Ackerly were available to the authors. The two general psychology evaluations from the final years of JP's life consisted of descriptions of observations rather than test scores. We were not able to track down a written report of JP's post-mortem brain examination, and we could not locate the preserved brain to study ourselves. However, all of the data uncovered by the authors were strikingly consistent with the original descriptions by Ackerly and Benton.

5. Conclusion

The case of JP, presented in 1947 by Ackerly and Benton at the Association for Research in Nervous and Mental Disease (Ackerly & Benton, 1948) continues to teach about the life trajectory that follows developmental frontal damage. The post-mortem brain examination reveals that JP suffered some damage to memory-related structures in addition to massive destruction of his prefrontal lobes and that he developed neurocognitive degeneration later in life related to hydrocephalus. In addition, JP's case illustrates, perhaps better than any other in the medical literature, that early life frontal damage causes persistent, life-long deficits in social adaptation and social cognition.

JP's case is unusual in that Ackerly followed his patient for the rest of JP's life. This longitudinal follow up included additional history that Ackerly published in 1964. We now also know that Ackerly arranged for a brain cutting and a photographic record of JP's brain for further study. Arthur Benton also continued to reflect on the lessons of this case later in his career (Benton, 1991, 2003). With the addition of the data in the current paper, the case of JP constitutes the longest recorded followup of a case of early childhood frontal injury.

It also provides a window into the unusual compassion and dedication of the outstanding clinician scientist, Spafford Ackerly.

It is rare in medical literature to find such a well-studied case that both correlates a constellation of behaviors richly described with focal brain dysfunction, and that also provides evidence for study by future physicians. For this reason, the case of JP, along with patients such as Phineas Gage, Victor LeBorgne, Auguste Deter, Solomon Shereshevsky, and Henry Molaison, can be considered a landmark case in the history of neuropsychiatry (Benjamin et al., 2018).

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Declaration of competing interest

Dr. Benjamin and Dr. Lauterbach are authors for and partners in Brain Educators, LLC, publishers of a neuropsychiatric examination pocket card. The other authors have no interests to declare.

Data availability

No data was used for the research described in the article.

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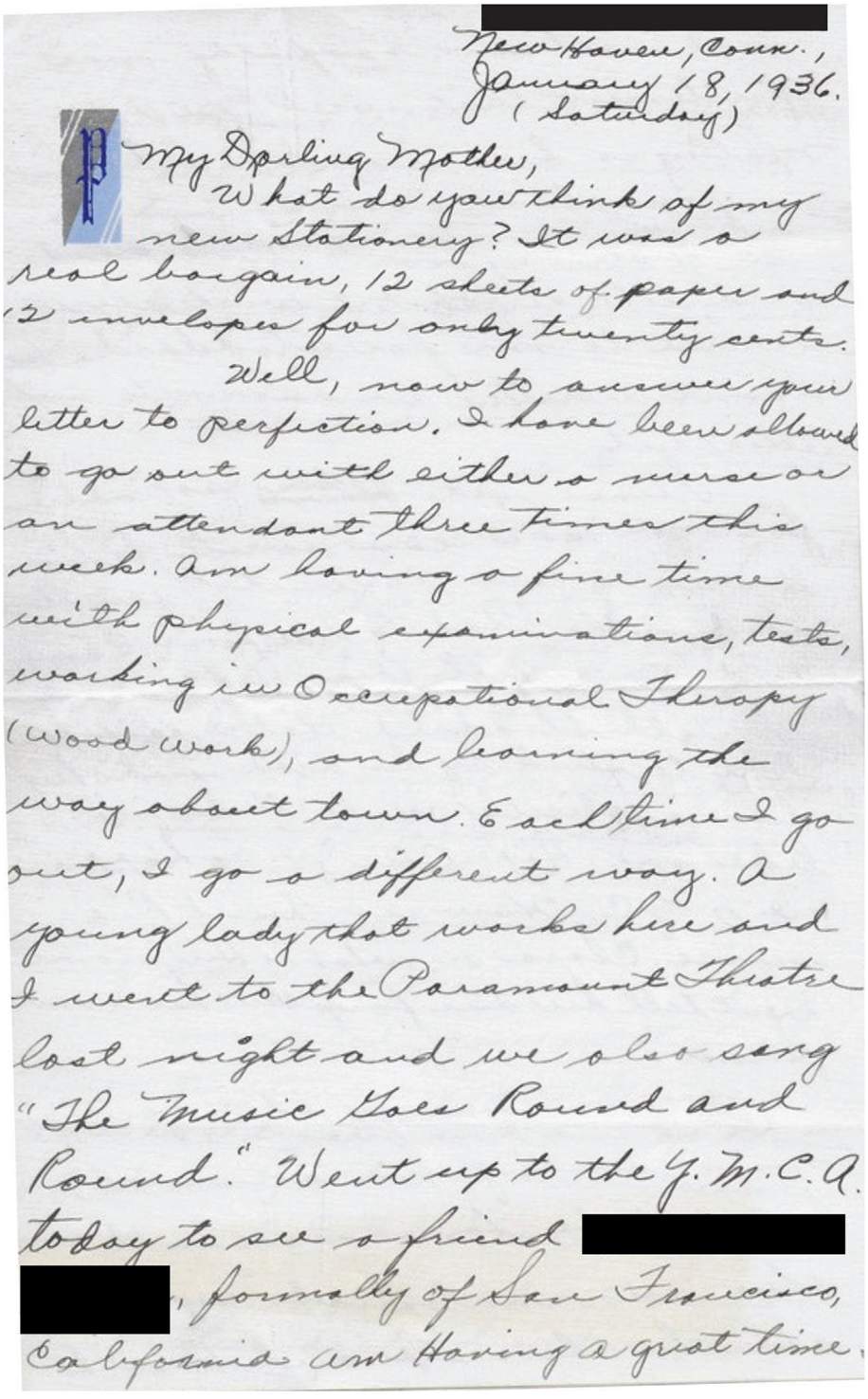
Appendix 1. Letter from JP to his mother dated January 18, 1936. This letter was written during JP's admission to Yale for evaluation. Krank's refers to a brand of brushless shaving cream. The names of the patient, family, and friends have been redacted.

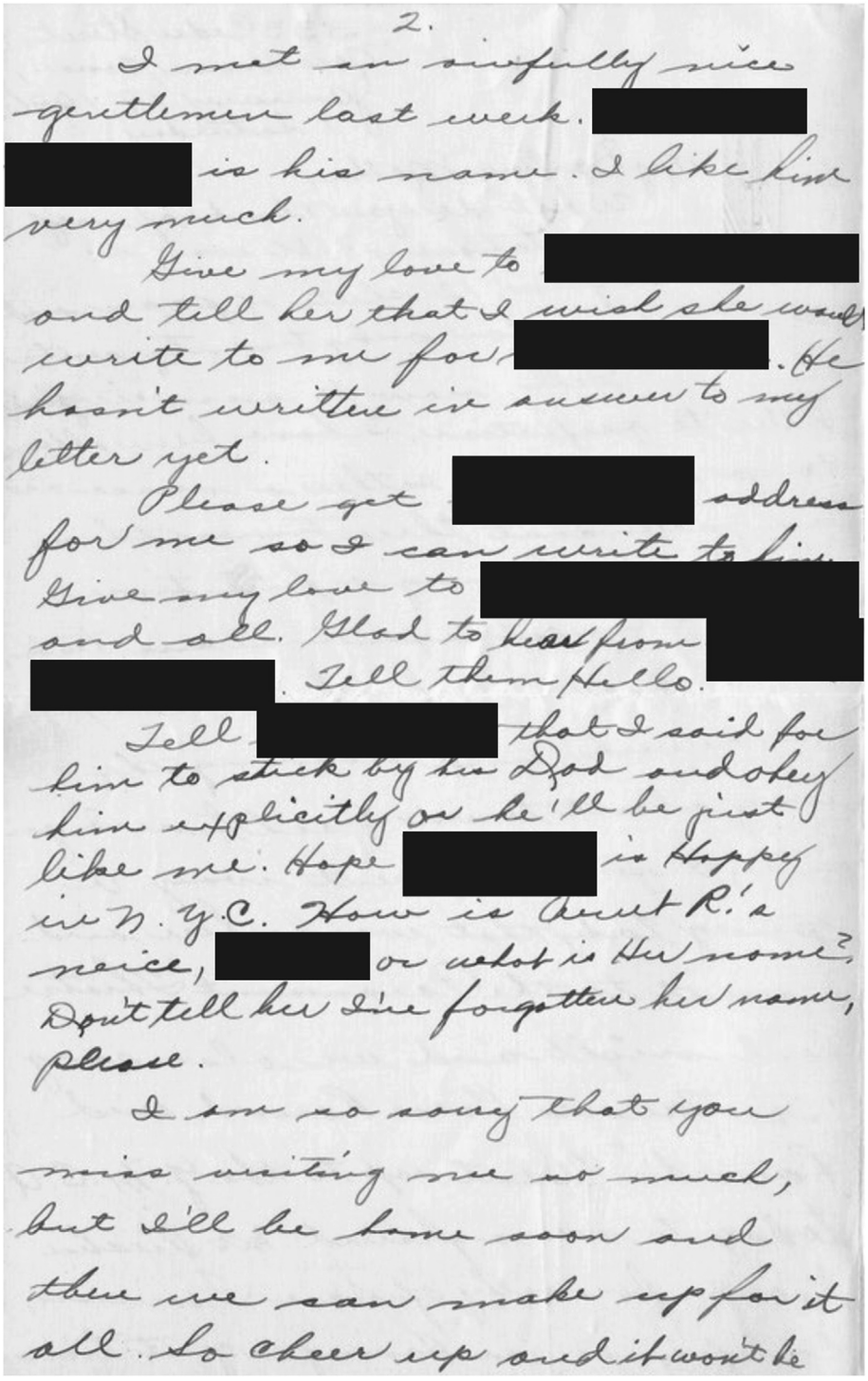


[redacted]
New Haven, Conn.,
January 18, 1936.
(Saturday)

My Darling Mother,

What do you think of my new Stationery? It was a real bargain, 12 sheets of paper and 12 envelopes for only twenty cents.

Well, now to answer your letter to perfection. I have been allowed to go out with either a nurse or an attendant three times this week. Am having a fine time with physical examinations, tests, working in Occupational Therapy (Wood work), and learning the way about town. Each time I go out, I go a different way. A young lady that works here and I went to the Paramount Theatre last night and we also sang "The Music Goes Round and Round." Went up to the Y. M. C. A. today to see a friend [redacted], formally of San Francisco, California am Having a great time.



2.

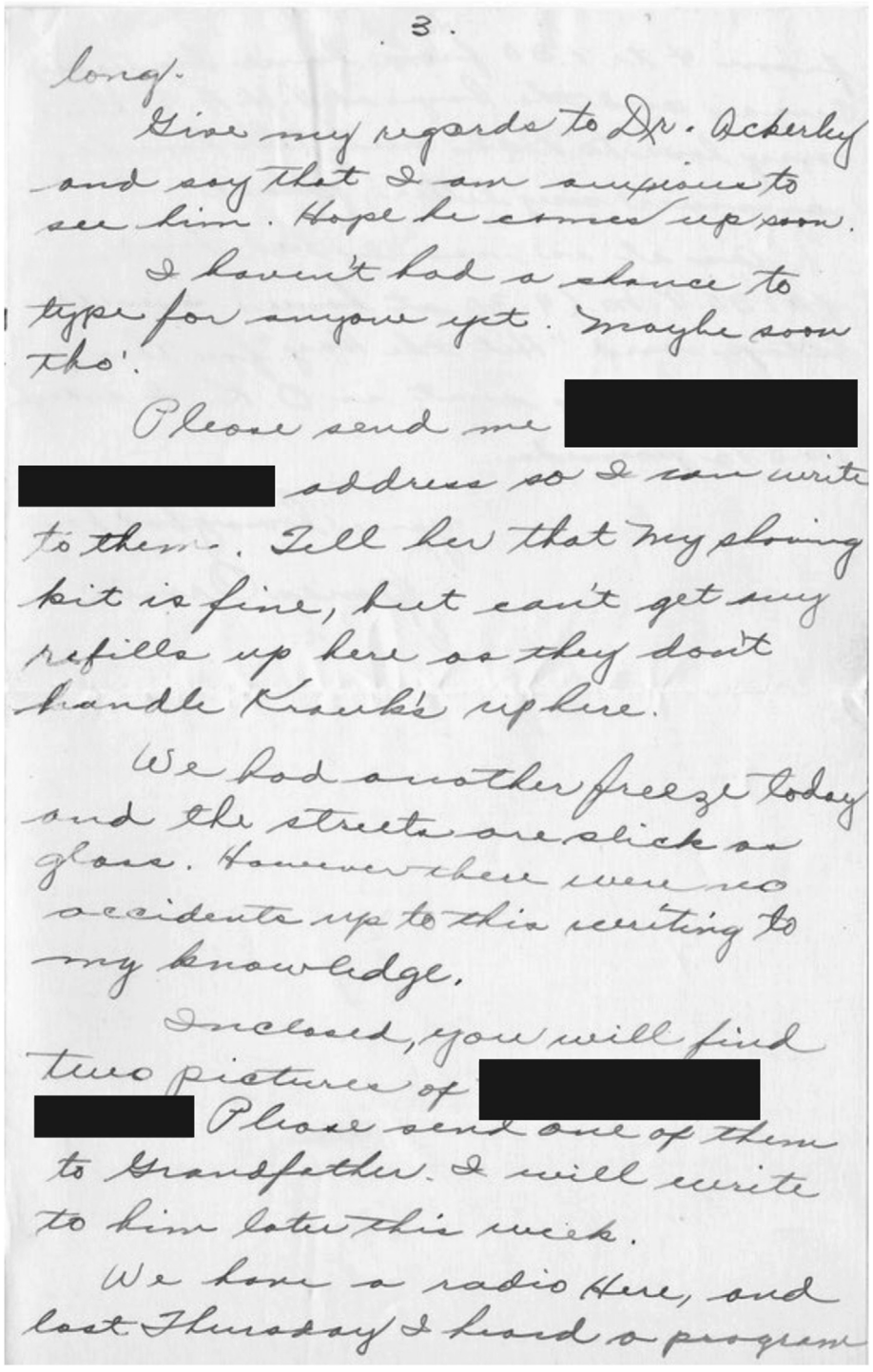
I met an awfully nice gentleman last week. [redacted] is his name. I like him very much.

Give my love to [redacted] and tell her that I wish she would write to me for [redacted]. He hasn't written in answer to my letter yet.

Please get [redacted] address for me so I can write to him. Give my love to [redacted] and all. Glad to hear from [redacted]. Tell them Hello.

Tell [redacted] that I said for him to stick by his Dad and obey him explicitly or he'll be just like me. Hope [redacted] is Happy in N. Y. C. How is Aunt R.'s neice, [redacted] or what is her name? Don't tell her I've forgotten her name, please.

I am so sorry that you miss writing me so much, but I'll be home soon and then we can make up for it all. So cheer up and it won't be



3.

long.

Give my regards to Dr. Ackerly and say that I am anxious to see him. Hope he comes up soon.

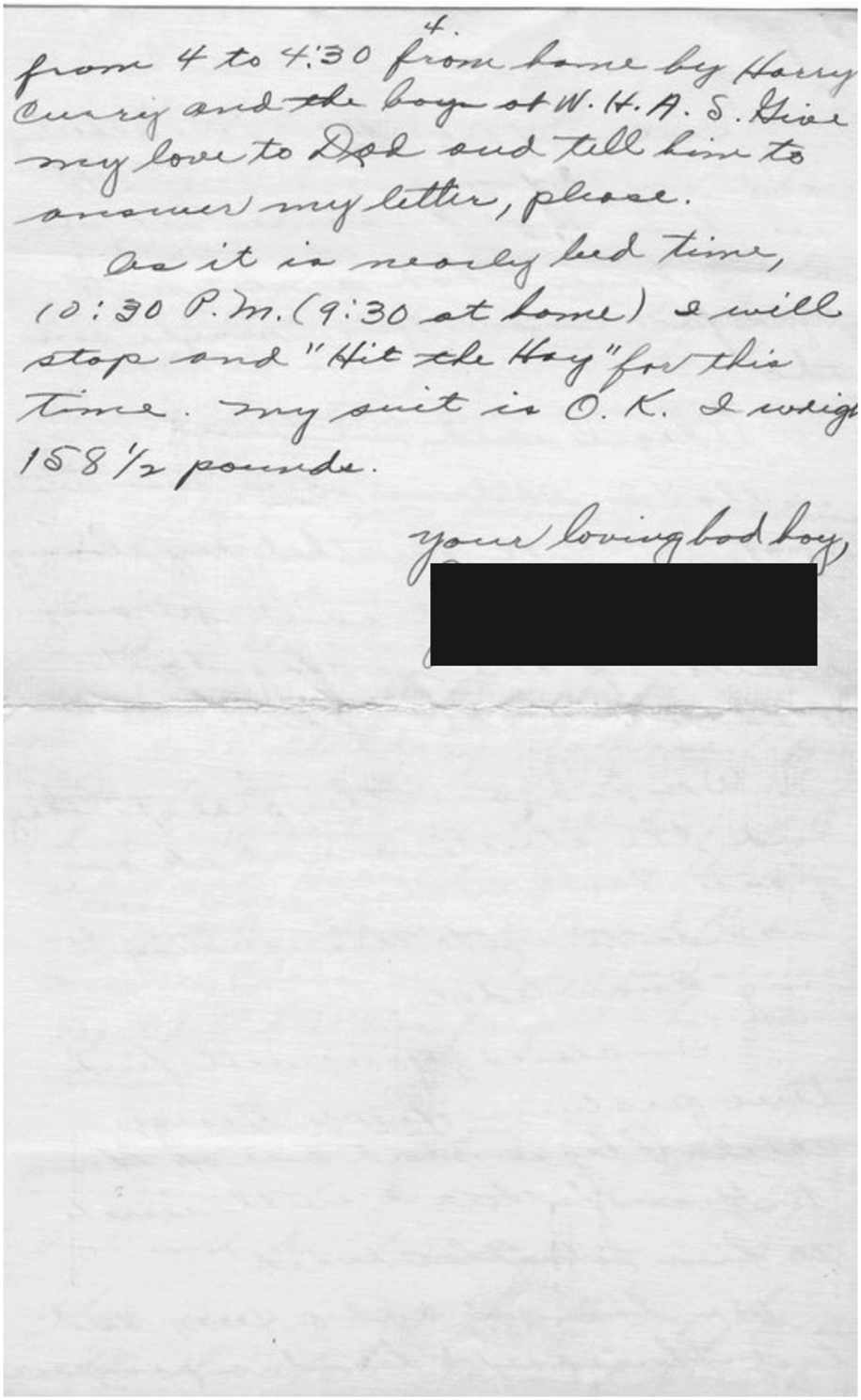
I haven't had a chance to type for anyone yet. Maybe soon tho'.

Please send me [redacted] address so I can write to them. Tell her that my sewing kit is fine, but can't get any refills up here as they don't handle Krank's up here.

We had another freeze today and the streets are slick as glass. However there were no accidents up to this writing to my knowledge.

Inclosed, you will find two pictures of [redacted] Please send one of them to Grandfather. I will write to him later this week.

We have a radio Here, and last Thursday I heard a program



4.

from 4 to 4:30 from home by Harry Curry and ~~the~~ boys at W. H. A. S. Give my love to Dad and tell him to answer my letter, please.

As it is nearly bed time, 10:30 P. M. (9:30 at home) I will stop and "Hit the Hay" for this time. My suit is O. K. I weigh 158 ½ pounds.

your loving bad boy,

[redacted]

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