

G YRATIONS

THE OFFICIAL NEWSLETTER OF THE AMERICAN SOCIETY OF PEDIATRIC NEURORADIOLOGY

MESSAGE FROM THE PRESIDENT

A DISCUSSION OF THE NEW SELF-ASSESSMENT MODULES (SAMS)

BY GARY L. HEDLUND, D.O.

The American Board of Radiology (ABR) Maintenance of Certification (MOC) program emphasizes quality improvement. Self-Assessment Modules (SAMs) are integral to life-long learning. SAMs are intended to extend the practitioner's habit of self-assessment, and is a vital component of the MOC process. SAMs are educational venues (e.g. refresher courses, focus sessions, workshops, reading assignments, etc) and are ABR-qualified. CME credit is also earned when a SAM is completed.

How many SAMs do I need and where will I get them?

Physicians who have obtained their diagnostic radiology certification by the ABR prior to June 2002 are strongly encouraged to enter into the MOC process. Individuals who were certified by the ABR at or after June 2002 are required to enter into the MOC program. The practitioner who holds a diagnostic radiology certificate irrespective of its issue date, and also holds a Certificate of Added Qualification (CAQ), is required to enter into the MOC process.

A diplomat who is enrolled in the MOC process will be required to complete 20 SAMs over a 10-year MOC cycle. The ABR advises that approximately 2 SAMs per year be completed. There are two SAM categories: general content and clinical content. The former represents areas of radiology practice that have significant impact on clinical practice. These SAMs are ABR prescribed. Examples of general content SAMs include: patient safety and radiation protection,

basic life support, advanced life support, and MRI safety. Four general content SAMs are required for all diplomats in the 10-year MOC cycle. Clinical content SAMs are diplomat selected, and should reflect the physician's current and/or anticipated practice environment. These clinical content SAMs include: musculoskeletal, neuroradiology, vascular and interventional, ultrasound, pediatric radiology, and breast imaging, just to mention a few.

If the diplomat holds only an ABR diagnostic radiology certificate, 16 clinical SAMs are required, in addition to the 4 general content SAMs. If the practitioner holds an ABR diagnostic radiology certificate plus one CAQ, 10 clinical content SAMs and 6 subspecialty clinical content SAMs (both diplomat selected) are required. If the practitioner holds two CAQs and an ABR diagnostic radiology certificate, the SAM requirements include 4 general content, 4 clinical content, 6 subspecialty content SAMs for the first CAQ, and 6 subspecialty clinical content SAMs for the second CAQ.

In summary, no matter what the diagnostic radiology/CAQ combination, 20 SAMs are required for the 10-year MOC cycle. See www.theabr.org for MOC requirements.

Are any SAMs currently available?

There are qualified and provisionally qualified SAM modules that have been developed by ARRS, RSNA, and ASNR. There are 5 pediatric neuroradiology SAMs that have been developed

Message From the President continued on page 7



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EDITOR'S NOTE:

Welcome to the first issue of *Gyrations*, the new ASPNR newsletter. We intend this publication to provide more than just Society news. We hope to address areas of controversy, provide relevant information about products and imaging techniques, and have fun in the process. We would appreciate feedback about the newsletter, good or bad, and are open to suggestions for articles and editorials. So take a look, and let us know your thoughts. Send your comments and suggestions via e-mail to the ASPNR Headquarters Office (bmack@asn.org). Please be sure to indicate "ASPNR Newsletter Comments" in the subject area of your e-mail.

IN THIS ISSUE:

Message from the President	1
What's Going on in Pediatric Neuroradiology Research?	2
The Retzius Neuroanatomy Quiz	3
Origin of the Term "Medulloblastoma"	4
The Art of Neuroradiology	8

ASPNR REMINDERS

Don't forget to pay your 2006 membership dues! If you need another invoice, contact the Headquarters office (bmack@asnr.org).

Know a colleague who might be interested in ASPNR membership? Applications for 2007 are available on our website (www.aspnr.org) or by contacting the Headquarters Office (bmack@asnr.org). The deadline to apply is October 10, 2006.

WHAT'S GOING ON IN PEDIATRIC NEURORADIOLOGY RESEARCH?

A Discussion of the Neuroimaging Center of the Pediatric Brain Tumor Consortium

By Tina Young Poussaint, M.D.

Director, PBTC Neuroimaging Center—Children's Hospital Boston
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The Neuroimaging Center (NIC) of the Pediatric Brain Tumor Consortium (PBTC), located at Children's Hospital Boston, is the first funded NCI-supported cooperative imaging center in the United States. Its mission is to provide leadership in diagnostic imaging within the PBTC therapeutic protocols. The PBTC is devoted to correlative tumor biology and new therapies for children with brain tumors. The imaging studies included are conventional MR, MR diffusion, MR perfusion, MR spectroscopy and PET.

The PBTC consists of 10 member institutions in the US. Each member site has a designated pediatric neuroradiologist who constitute the Neuroimaging Committee. The role of this committee is to collaborate with the NIC regarding quality assurance procedures, protocols, and research projects for MR. Meetings are held twice a year at the biannual PBTC meetings, with interim communication by e-mail or phone. For PET QA and protocols, a PET investigator committee composed of a PET investigator from each site provides input about quality assurance procedures, protocols, and research studies. A group of individuals at Children's Hospital Boston with expertise in clinical research, collaboration, data analysis of MR and PET, quality assurance (QA), information technology and transfer constitute the research team.

The NIC works closely with the PBTC Operations and Biostatistics Center (OBC), the PBTC scientific leadership to foster the incorporation of key imaging objectives in developing protocols. All imaging data is submitted by electronic data transfer that occurs across a HIPAA compliant secure tunnel from the participating sites to the OBC at St. Jude's Children's Research Hospital. A quality assurance system for verifying the components of the required imaging studies is used. PET data is forwarded to the OBC by tunnel download from the site or by FTP. Image fusion of the MR exams with PET is done using a multimodality processing and review station (Hermes Medical Solutions).

A quality-assurance program is in place for both MR and PET. The MR QA program utilizes quarterly scanning of the ACR phantom. The PET QA process is a quarterly evaluation form completed by each site with a one-time scanning of a designated PET phantom by each site. The quality assurance program involves data and equipment integrity and compliance with protocols.

Image analysis is done with the help of a dedicated team which includes pediatric neuroradiol-

ogists, MR and nuclear medicine physicists, data analysis technologists, and information technology specialists. Efforts are made to develop quantitative parameters from the imaging components performed. The Clinical Research Program is used for a dedicated study coordinator, for data entry into the tunnel at OBC, and for the creation of forms and the procedure manual. The OBC is involved in all aspects of statistical design and analysis of the imaging data analyzed at the imaging center.

Neuroimaging research questions are incorporated into the therapeutic aims of the PBTC protocols. The incorporation of the appropriate imaging parameters into the PBTC protocols is based upon the input of the Neuroimaging and PET Investigator Committees. In one protocol, PBTC-002, a Phase 1 study of an antiangiogenesis drug called Semoxind (a novel small molecule inhibitor of vascular endothelial growth factor), volumetric MR, MR diffusion, MR spectroscopy, and MR perfusion were incorporated as required imaging studies. As has been shown in adult studies and animal models, diffusion was increased in the tumors of long-term survivors and decreased in the short-term survivors. The increased diffusion possibly reflects increased apoptosis and decreased cellularity in the tumor consistent with tumor response to therapy. Additional data from other PBTC studies with similar tumor types and treatments will be pooled to determine if there are significant findings demonstrated in other imaging parameters over time. In one antiangiogenesis study in development, the comparison between T2* perfusion and permeability perfusion imaging will occur to determine if there are early changes in perfusion that can effect treatment response and, potentially, determine further drug dosing. Preliminary data obtained in pooling permeability studies from two of the PBTC protocols demonstrated an increase in permeability from baseline to follow up, which was greater in progressive patients than stable patients. Such trends will need further statistical validation in future trials.

Further research evaluation and validation of the neuroimaging tools used to evaluate the child with brain tumor is needed to understand current and future techniques that can serve as imaging markers useful for diagnosis, treatment, response, and evaluation of neurotoxicity, adding to further metabolic and physiologic characterization of brain tumors.

THE RETZIUS NEUROANATOMY QUIZ

The Retzius neuroanatomy competition is an annual event that has been going on for over 10 years. The competition takes place in Los Angeles the first week in April. The competition consists of 60 questions of normal neuroanatomy in which the contestants are asked to name structures pointed out on whole brain sections, intra-op photos, angiograms, MRs, CTs, etc. The competition is open to all fellows, residents of any specialty, and medical and graduate students. There is no entry fee. The winner gets \$1,000 and a special bronze medal sculpted by the famous medal artist Alex Shagin. Second place is \$500, third place \$250. Gustav Retzius (1842-1919) was a noted Swedish anatomist and histologist at the

Karolinska Institute, and was one of the leaders in research during the classical period of neuroanatomy. "His comparative studies of a large series of subprimate, simian, and human brains, fetal and adult, clarified many of the more difficult problems of brain morphology." (Haymaker-Schiller, Founders of Neurology.)

This column will highlight points of neuroanatomy, first with a quiz and then a description of the structure named and clinical significance of lesions involving the structure, when possible.

Anyone interested in entering this competition should contact Dr. Marvin Nelson (mdnelson@chla.usc.edu).

KEY DEFINITIONS

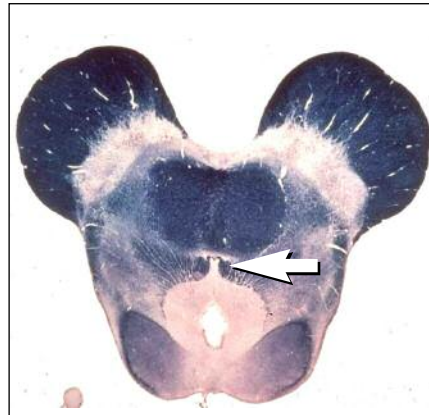
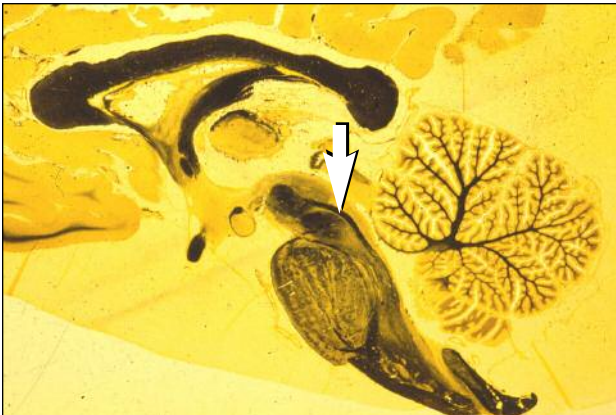
Fasciculus: fibers that run together which originate from multiple different nuclei and have multiple different endpoints. For example; pyramidal fasciculus (not tract).

Tract: fibers that run together which originate from a common group of nuclei and have a common endpoint. For example; corticospinal tract, mamillothalamic tract.

Lemniscus: a general term for a band or bundle of nerve fibers in the central nervous system.

DISCUSSION OF THE RETZIUS

Identify the structure below (arrow)



ANSWER TO RETZIUS
NEUROANATOMY QUIZ
ON PAGE 8

THE ART OF NEURORADIOLOGY

This column will present all forms of art and literature pertaining (however obliquely) to neuroradiology. This may include photographs, drawings, poems, limericks, etc.

As an example, who is depicted in this work?
(Hint: it was done 20 years ago.)



ORIGIN OF THE TERM “MEDULLOBLASTOMA”

By Floyd Gilles, M.D.



Figure 1 Wurffbain's drawing of a 2 year old with hydrocephalus and a posterior fossa tumor (1691). The nature of the tumor is unknown.¹

We all learn so many different terms for disease processes during our training. Many terms have become obsolete, but continue to be used and mixed in with new terms. The following article is an interesting story about the origin of the term “medulloblastoma.” The article is by Dr. Floyd Gilles, the Burton E. Green Professor of Pediatric Neuropathology at Children’s Hospital Los Angeles/USC.

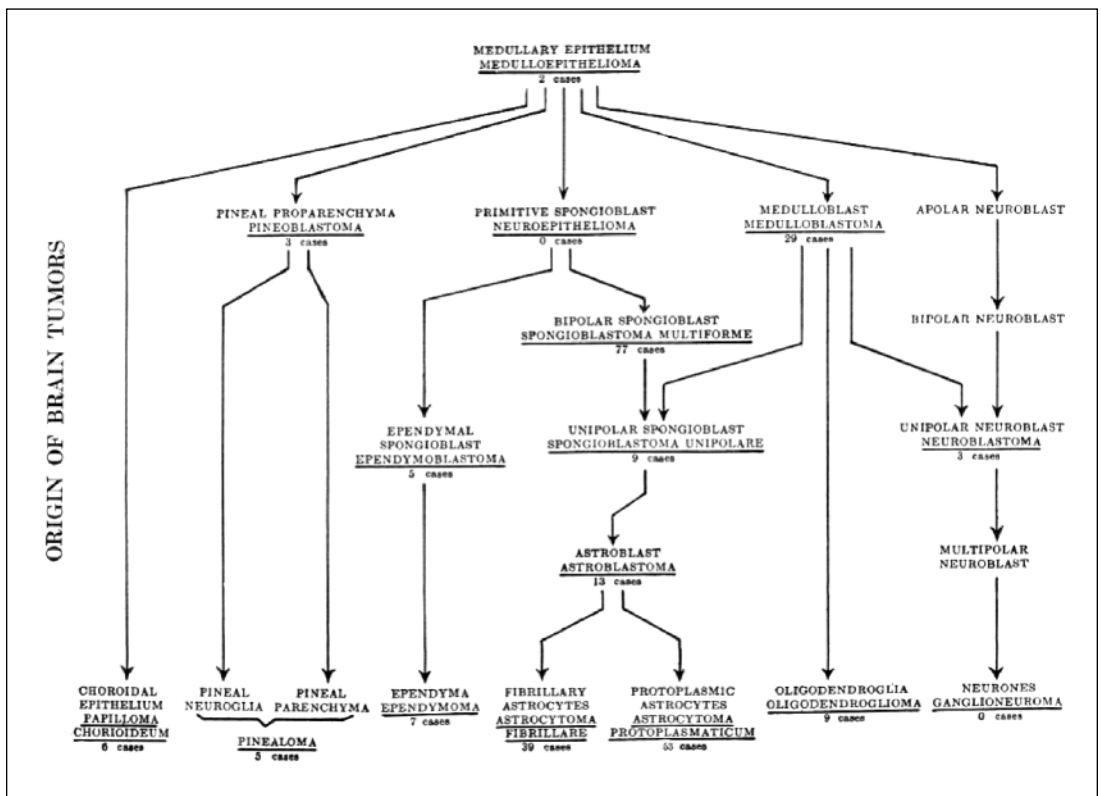
Perhaps the first description of an infant with a posterior fossa tumor is that of Wurffbain in 1691 (Figure 1).¹ During the late 1800s, the soft posterior fossa tumor composed of small to medium sized hyperchromic cells was generally called a sarcoma, as were the other neuroglial tumors. As cited by Bailey, Bucy, and Buchanan, Ollivier described a childhood densely cellular midline cerebellar tumor in 1827^{3,4} (before cell theory was established in 1838⁵) as a “sarcoma,” a densely cellular tumor. Hughlings Jackson, a half-century later (1881), described a midline cerebellar tumor as being composed of a multitude of “corpuscles” (well after cell theory was established).⁶

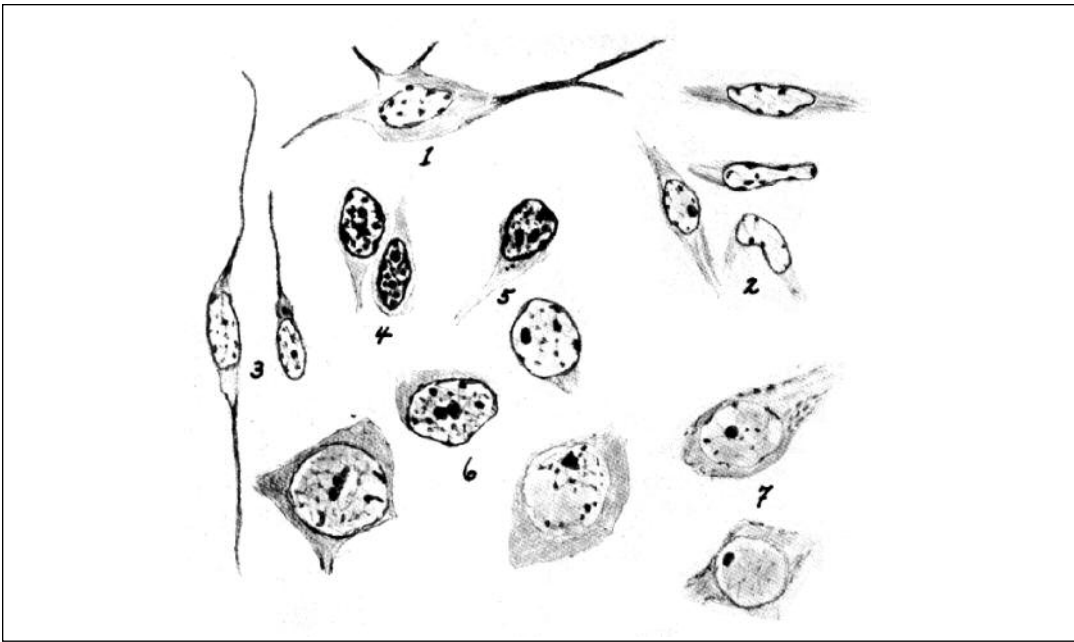
Percival Bailey and Harvey Cushing undertook in the early 1920s to form a new classification of 400 “verifiable gliomas.” “The peculiar life history of a particular tumor of any sort becomes classified; and without such clarification the bedside diagnosis must remain obscure, the preferential

treatment cannot be standardized, and the prognosis will be wholly a matter of guesswork.” This classification was based on nervous system histogenesis, the different tumor groups being named according to the predominating embryological type of cell composing the tumor.² They challenged the then current belief “the microscopical examination of a specimen removed at operation will not serve to predict, with any degree of certainty, the future course of development of a true tumor of the brain substance.” Störch had, two decades earlier, issued the same challenge when he recognized in 1899 that a new tumor, the ependymoma, had sharp edges and should be removable surgically.⁷

Bailey and Cushing accepted the dogma of Ribbert (1918) that brain tumor cells were derived from embryonic cells important in brain development. Ribbert, in turn, had accepted Cohnheim’s hypothesis that tumors arose from cell rests or superfluous groups of cells which, during development, failed to mature; but persisted in their embryonic state,^{8,9} even though contemporaneously the hypothesis was soundly refuted.¹⁰ Ribbert’s 1918 claim that brain tumor cells recapitulated different stages of neural cell development became the conceptual basis for the 1926 Bailey and Cushing histogenetic classification of 400 brain tumors (Figure 2).¹¹

Figure 2 Bailey and Cushing’s Histogenetic Classification.¹¹





1. Neuroglial cell
2. Four cells of the reticulum of the connective tissue
3. Group of two spongioblasts, one bipolar, one unipolar
4. Two medulloblasts
5. A medulloblast
6. Group of four neuroblasts
7. Two neuroblasts (From Bailey²)

In 1924, Bailey and Cushing took to the American Neurological Association annual meeting a new small cell midline tumor in the posterior fossa in children under the name spongioblastoma cerebelli. The preceding paper was that of Strauss and Globus, who had previously used the same term "spongioblastoma" (apparently independently of Ribbert) for a cerebral tumor in adults with intense vascularity, extensive necrosis, hemorrhagic foci, and cystic degenerative changes and that giant cells were common¹². Strauss and Globus called their tumor "spongioblastoma multiforme." Some of Bailey and Cushing's tumors had been previously designated neurocytoma or neuroblastoma, terms introduced earlier by Homer Wright for a suprarenal tumor arising from the sympathetic nervous system, or a tumor related to retinal gliomas.¹³⁻¹⁵ Both the cerebellar and suprarenal tumors sometimes had rosettes and tended to occur in children.

There was considerable discussion between these two groups of investigators. Bailey and Cushing agreed that the tumor cell nature was not evident microscopically. They thought they were related to the indifferent cells of Schaper¹⁶ or "...as we prefer to call them, medulloblasts." But where did the term "medulloblast" arise? Certainly, Bailey and Cushing did not use this term,¹⁷ when they first presented their paper to the American Neurological Association. In the discussion after the presentation, they allowed that Drs. Globus and Straus had priority to the name "spongioblastoma" because of their earlier case report in 1918. Harvey Cushing, addressing the audience during the discussion of these papers stated "It is futile, I think, to inflict on the Society any further discussion in this matter, and I propose that Dr. Globus and Dr. Straus, Dr. Bailey and I foregather in some quiet antechamber and thrash this matter out together, rather than to take

the Society's time by doing so in open meeting." It was in that antechamber that the name "medulloblastoma" was conceived, derived from a new cell postulated important in nervous system histogenesis. The tumor name has subsequently been fossilized into our nomenclature.

Bailey and Cushing initially had 25 cases. Subsequently, they added 4 cases to their series.¹⁸ They decided that in all 29 cases the neoplasm originated from the fourth ventricular roof, and that it developed as a central intracerebellar lesion. Ten per cent of Cushing's cases spread in the leptomeninges and contained an unusual amount of connective tissue. As Bailey and Cushing developed their series, they began to use radiation in the postoperative period, and found that survival improved. Several of Bailey and Cushing's cases were tumors in the cerebrum, but they later recanted and said such tumors only developed in the midline cerebellar region, primarily in children.¹⁹ Others, finding medulloblastoma-like tumors elsewhere in the nervous system, gave them various names. Cushing later stressed the later age incidence and slower growth of cerebral compared to cerebellar medulloblastoma.²⁰ Later, Bailey also changed his mind and allowed that occasionally these tumors could be found in the cerebrum or spinal cord.²

The "medulloblast" has not been accepted as a precursor cell of neural cells. Marburg in 1931 and, later, Stevenson and Echlin in 1934 postulated the origin of medulloblastoma from persistence of the cerebellar external granule cell layer postnatally, because they found subpial small cells.^{21,22} This position was still taken in 1970 by Kadin, Rubinstein, and Nelson.²³ However, one typical growth pattern of medulloblastoma is subpial growth, which simulates external granule cell

Medulloblastoma continued on page 6

layer persistence. Originally, it was thought that these cells could also give origin to neuroglia as well as neurons,²⁴ but the external granular cells are already committed as neuroblasts before migration from the subventricular zone in the fourth ventricular roof.^{25,26} Kershman failed to find medulloblasts in the cerebrum and spinal cord, only in the cerebellar external granule cell layer²⁴; subsequently, subpial neuroblasts were found in the cerebrum.^{27,28}

On the other hand, Raaf and Kernohan ascribed the origin of medulloblastomas to abnormal collections of small cells (rests) in the posterior medullary velum.²⁹ These small cells were intermingled with ganglion cells. They used 161 embryos, fetuses, and infants and found that the rests were different from the germinal bud that normally disappears by one month of age. Twenty-three of 104 cerebellums containing the posterior medullary velum contained these collections. Twelve of the 23 infants or fetuses with these abnormal collections were past the age at which the germinal bud disappears. One such collection was found in a 24-year-old adult without a medulloblastoma, but with a pinealoma. They are distinct from the heterotopias found elsewhere in the cerebellum.³⁰ They thought these posterior medullary velum collections were abnormal, because their morphologic appearance and arrangement were not those of the normal germinal bud. In one cerebellum the appearance was that of a tumor. The cells simulated those seen in medulloblastoma and contained many mitoses and pseudorosettes and occasional neurons, which they interpreted as Purkinje cells. The trouble with this argument, is that some classic medulloblastomas are located in the cerebellar hemisphere.³¹ Abbott and Kernohan suggested that medulloblastomas be graded 1-4,³² but currently all are considered grade IV.³³

Tumor in leptomeninges elicits desmoplasia, but reticulin deposition around tumor cells is different. Bailey and Cushing recognized desmoplasia in three of their medulloblastomas. They felt that this resulted from invasion of meninges and cited Ollivier's case in 1837, as well as 17 additional cases of cerebellar medulloblastomas from the literature prior to their publication. In 1927, Bailey revived the question of primary sarcomas.³⁴ In some medulloblastomas, reticulin appears to have been produced by the tumor cell; at least each tumor cell is surrounded by reticulin. Twelve years later, Bailey, Bucy and Buchanan presented 19 cases of malignant midline cerebellar tumors in children,⁴ six of which were considered sarcomas, because they contained reticulin. They felt that these occurred in an older group of patients and were more slowly growing. Some tumors in the leptomeninges were not associated with vermal tumors, but arose in the hemisphere.

In 1962, Kernohan and Uihlein presented the argument for an entity they called circumscribed

sarcoma of the cerebellum.³⁵ Unlike the soft gelatinous nature of medulloblastoma, the circumscribed sarcoma of the cerebellum was firm or hard. They denied the usefulness of the reticulin stain, because medulloblastoma cells stimulate the leptomeninges to produce reticulin. The tumor cells of circumscribed sarcomas were larger, had more cytoplasm, larger nuclei, and less dense chromatin. They found that these tumors characteristically had "glomeruli" or islands of more loosely packed cells in the midst of dense cellularity.³⁶ The glomeruli did not contain reticulin, but contained neoplastic cells larger than those in the surrounding tumor. Rubinstein and Northfield demolished this argument in a review in 1964.³⁷ In 1965, Dexter and Howell resurrected the desmoplastic medulloblastoma³¹ and in 1971, Chatty and Earle provided a description similar to that of Kernohan: "The tumor is made of islands of cells entirely devoid of connective tissue, surrounded by and delineated from the rest of the tumor by an elaborate network of fibrous tissue rich with reticulin." They thought that prognosis was improved in the desmoplastic medulloblastoma, but did not properly correct for age.³⁸ The current WHO definition is similar: "This lesion shows nodular, reticulin-free zones surrounded by densely packed, highly proliferative cells that produce a dense intercellular reticulin fiber network".³³

Posterior fossa small-celled densely cellular tumors have been known for a long time under at least two designations. Their history is filled with arbitrary names and limited characterizations of defining histologic features. Today, even though we no longer grade medulloblastomas, there may be multiple subsets.³⁹

Floyd Gilles is the Burton E. Green Professor of Neuropathology at Childrens Hospital Los Angeles and is a Professor of Neurology and Pathology at the USC Keck School of Medicine.

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Message from the President continued from page 1

by either the RSNA and/or the ARRS. These can be accessed through the education portals of the institution's main web page. A list of all SAM modules can be viewed at www.theabr.org.

What are the components of a SAM?

Each SAM educational event must be at least 90 minutes in length, and be ACGME approved for 1.5 hours of category I physician credit. Each SAM session will conclude with a minimum of 5 referenced multiple-choice questions having four or five selections and one correct answer. From the SAM educational event, the ABR desires that the participant will have rapid personal feedback, and the organization will receive: (1) "Scored distributions," which represent the participant's scores on the post-test assessment and, (2) the distribution of selected answers by each participant. These data can be in the form of a Table or Histogram. Once a SAM has been qualified by the ABR, it will have a lifetime of three years. The submitting organization may petition to reactivate the SAM as is, or modify it for an additional three-year cycle. For previously published images used within a SAM, the Society or Journal holds the copyright and the ABR would need to seek permission to use previously published images.

What relevance does the SAM have to my current or future practice?

The SAMs that a physician completes in the 10-year MOC cycle will be used to shape the content of the cognitive expertise computer-based examination. The ABR plans to begin a review of the completed SAMs at approximately the seventh year of the MOC cycle.

Does the ASPNR have plans to integrate SAMs into upcoming meetings?

Two SAM educational modules will be offered during the ASPNR programming (May 1-2, 2006) at the upcoming ASNR 44th Annual Meeting in San Diego. SAM and CME credit will be available for members attending the *CT Dose Reduction: Current Practices in Pediatric Neuroradiology* and the *Pediatric Orbit and Temporal Bone Imaging* sessions. Both of these sessions will be equipped with audience response systems to facilitate a

smooth accumulation of response data that will satisfy the SAM requirements by the ABR.

The current ASPNR plan for pediatric neuroradiology SAM development is to begin by having the Executive Committee, led by the ASPNR President, integrate SAM-qualified sessions into ASPNR programming during the ASNR Annual Meeting. These sessions will also become part of a durable ASNR electronic repository available for member use. Beginning now, and in the future, I see a representative of the ASPNR Executive Committee working with the ASNR Electronic Education Committee in developing a system for archiving pediatric neuroradiology SAMs. I would expect interested members of the ASPNR to contribute to these modules. Outlining and monitoring the content of pediatric neuroradiology SAMs will, in the foreseeable future, be the responsibility of the ASPNR Executive Committee.

How do I keep track of my SAM credits?

Organizations such as the RSNA and ARRS have made inroads into tabulating and transferring SAM participation by members to the ABR through a "gateway" interface, which acts as a conduit for both CME and SAM credits. The ASNR, as of the writing of this newsletter, has made no such agreements. No later than January 2007, diplomats of the ABR may individually access and report their CME and SAM credits within the MOC personal profile portal of the ABR website. At this time, the diplomat should maintain a paper trail of SAM and CME credits.

Who is coordinating pediatric neuroradiology SAMs among the various radiology organizations?

Currently, there has been no coordination between ARRS, RSNA, SPR, and ASNR (ASPNR) when it comes to SAM development of pediatric neuroradiology content. The ASPNR President and Vice-President/President-Elect will be working together to direct SAM content for future ASPNR meetings.

If you have further questions regarding your SAM requirements as a diplomat and ASPNR member, please see www.theabr.org.

MEETINGS OF INTEREST

NER Foundation 2006 Symposium & ASNR 44th Annual Meeting at the San Diego Convention Center (April 29-May 5, 2006). ASPNR programming will take place on May 1-2. Registration is now open at www.asnr.org.

2006 International Pediatric Radiology Meeting (5th Conjoint Meeting of the SPR and ESPR) at The Fairmont The Queen Elizabeth Hotel, in Montreal. For more information, visit pedrad.org.

NEXT ISSUE...

Is 3T MR ready for Pediatric Neuroradiologic studies?

A review of GE, Siemens, and Philips 3T MR systems

"The medial longitudinal fasciculus is a heavily myelinated composite tract, lying near the midline, ventral to the periaqueductal grey matter. It ascends to the interstitial nucleus (of Cajal) which lies in the lateral wall of the third ventricle, just above the cerebral aqueduct. The fasciculus retains its position relative to the central grey matter through the midbrain, pons, and upper medulla, but is displaced ventrally by successive decussations of the medial lemnisci and lateral corticospinal tracts. At spinal levels it is synonymous with the medial vestibulospinal tract. The medial longitudinal fasciculus interconnects the oculomotor, trochlear, abducens, Edinger-Westphal, vestibular, reticular and spinal accessory nuclei, coordinating conjugate eye movements and associated movements of the head and neck. All four vestibular nuclei contribute ascending fibres. Those from the superior nucleus remain uncrossed, while the others are partly crossed. Some

fibres reach the interstitial and posterior commissural nuclei, and some decussate to the contralateral nuclei. Descending axons, from the medial vestibular nuclei and perhaps the lateral and inferior nuclei, partially decussate and descend in the fasciculus as the medial vestibulospinal tract. Fibres join from the dorsal trapezoid, lateral lemniscal and posterior commissural nuclei, which means that both the cochlear and vestibular components of the vestibulocochlear nerve may influence movements of the eyes and head via the MLF. Some vestibular fibres may ascend in the MLF as far as the thalamus."

"Lesions of the MLF cause internuclear ophthalmoplegia."

— Gray's Anatomy. Standring, S. Editor-in-chief. 39th Edition. Elsevier, London, 2005

Medulloblastoma continued from page 6

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