Written Public Comments

IACC Full Committee Meeting

April 30, 2010

List of Written Public Comments

Note: Personally Identifiable Information (PII) has been in this document

Marian Dar

January 20, 2010

Subject: Miscellaneous

There have been articles recently about how across the country pediatric specialties are under-served; maybe there can be an *increased effort to expand and distribute these type of clinical services and supports e.g., developmental pediatricians and psychologists*, pediatric gastrointestinal (GI), etc.

RE: [PII redacted]

Question asked was -- why "international"

Information exchange/"best practices" e.g.: My son who was born and spent early childhood oversees participated in a terrific occupations therapy (OT) (sensory integration) program with a boy from Malaysia (completely non-verbal) that was offered by a Australasian group; though conceived in the United States (US) it was not supported well because -- "no data." ... (Legitimate reservation, but how much and how long do we wait -- what guidelines??)

There were physicians (Hopkins!, United Kingdom (UK), etc.) and other clinical/education professionals combining overseas education and local practice with distinctive and superior result; autism is a heterogeneous phenomenon that may respond and be related to something new and unexpected. At some point, there is marginal and consequential benefit to broad exposure and thinking, working deductively –

Thank you.
Marian Dar

Eileen Nicole Simon

January 25, 2010

Subject: Discussion points

I was very happy to hear that members of the IACC plan to discuss comments submitted by the public. Thank you. This should be an important part of the mission of the IACC. Attached is a document with some issues I would like to hear discussed. This is preliminary (or background) for inquiry into current "accepted practices" in obstetrics and neonatal care. I have also posted this document at: http://www.conradsimon.org/IACCdiscussionPointsJan2010.pdf

See also a brief listing below. Thanks.

Eileen Nicole Simon
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In the attached document I have provided references, tables, and figures in support of the following:

- Evidence that the auditory system of the brain has higher blood-flow and metabolism than any other area of the brain.
- Evidence that the auditory system is susceptible to damage by circulatory arrest. Evidence that the auditory system is susceptible to impairment by toxic substances.
- Evidence that the auditory system is susceptible to impairment by loss of aerobic enzyme function when the co-enzyme thiamine (vitamin B1) is lacking.
- Evidence that the auditory system is part of the subcortical pattern of damage found in children affected by kernicterus.
- Evidence that bilirubin staining of the basal ganglia and other subcortical areas is secondary to impairment of the blood-brain barrier.
- Evidence that synthetic vitamin K and sulfisoxazole antibiotic are toxic to the blood-brain barrier.
- Evidence that damage of subcortical sites in the perinatal period leads to disruption of normal maturation of the cerebral cortex.

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Conrad Simon Memorial Research Initiative
To seek understanding of brain system impairments in autism.
http://conradsimon.org/

Attachment #1 to Eileen Nicole Simon's January 25, 2010 Submission

Discussion points submitted to members of the IACC, 25 January 2010

Evidence that the auditory system of the brain has higher blood-flow and metabolism than any other area of the brain [1, 2]. See figures 1 and 2 below.

Note: The deoxyglucose method has been extensively used in animal research on brain activity responses to drugs and other factors. The inferior colliculus has consistently been found to have the highest rate of glucose utilization in every study.

Evidence that the auditory system is susceptible to damage by circulatory arrest [3-13]. See figures 3, 4, and 5 below.

Note: Asphyxia at birth was produced in monkeys by preventing the first breath and cutting off circulation to the placenta (by clamping the umbilical cord). Ischemic bilaterally symmetric damage to brainstem nuclei resulted.

Note: Current accepted practice in obstetrics is to clamp the umbilical cord immediately after birth, whether or not the first breath has occurred [14].

Evidence that the auditory system is susceptible to impairment by toxic substances [15-36].

Note: Toxic substances cause bilaterally symmetric hemorrhagic (petechial) lesions of brainstem nuclei (Wernicke's encephalopathy). The lesions are petechial, resulting from increased blood flow in response to the insult, with bursting of capillaries. Toxic substances impair aerobic metabolism.

Evidence that the auditory system is susceptible to impairment by loss of aerobic enzyme function when the co-enzyme thiamine (vitamin B1) is lacking [37-49]. See figures 6, 7, and 8 below.

Note: Beriberi was once believed to be caused by a bacterial infection: "The cause of beriberi appears to be a bacillus which gains entrance to the alimentary canal in contaminated food and drink." [50, page 289]

Evidence that the auditory system is part of the subcortical pattern of damage found in children affected by kernicterus [51, 52]. See figure 9 below.

Note: Ranck and Windle (1959) in their paper on asphyxiation of newborn monkeys commented: "The human neuropathologic entity most closely resembling the effects of asphyxia neonatorum in the monkey is kernicterus. There are similarities in the distribution and type of nerve cell changes in both conditions. Major differences between the findings in the monkey and those in human infants with kernicterus are absence in the former of the usual history of erythroblastosis fetalis, lack of clinical jaundice, lack of pigment in the lesions, . . ." [4, page 153]

Evidence that bilirubin staining of the basal ganglia and other subcortical areas is secondary to impairment of the blood-brain barrier [53-57].

Note: Bilirubin is not toxic. Bilirubin staining of subcortical nuclei results from impairment of the blood-brain barrier by anoxic-ischemic injury or by a substance that disrupts aerobic metabolism.

Evidence that synthetic vitamin K and sulffisoxazole antibiotic are toxic to the blood-brain barrier [58-67]

Note: Natural vitamin K is fat soluble. Synthetic versions are water soluble, thus could be given by injection. Robertson pointed out that vitamin K became the second standard treatment for all newborn infants: "The prophylactic use of vitamin K in newborns began in the early 1940s and was the second routine pharmacological treatment of newborn infants; the first being the use of silver nitrate to prevent ophthalmia neonatorum." [63, page 53]

Comment: Discovery of the toxicity of synthetic vitamin K occurred at the same time that autoradiography revealed high blood-flow and metabolism in the inferior colliculi and other subcortical structures affected in kernicterus. Why have these elegant studies of brain activity been so neglected? Vitamin K continues to be part of accepted practice even though its routine administration to all newborns continues to be controversial and rightfully so [62, 63].

Evidence that damage of subcortical sites in the perinatal period leads to disruption of normal maturation of the cerebral cortex [68]. Sites of injury found in monkeys subjected to asphyxia at birth are listed in table 1 below. Sites of secondary growth failure are listed in table 2.

Note: Faro and Windle examined the brains of monkeys kept alive for many months or years after being subjected to asphyxia at birth. They noted "structural changes sequential to the initial asphyxial lesions . . . It was difficult to separate the primary effects of asphyxia from these later atrophic changes in some regions . . ." [68, page 41].

"Some of these regions, such as the frontal and parietal cortex, will be recognized as locations normally receiving the terminations of tracts destroyed by the primary lesions. . . . Extensive primary damage by asphyxia at birth led to a reduction in amount of white matter, as in the corpus callosum . . . All the animals had severe brain damage, but some were worse off than others." [68, page 43]

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Table 1: Brainstem Sites Damaged by Asphyxia at Birth
Inferior colliculi and Superior olives
(acoustic processing & relay)
Trigeminal nerve sensory nuclei
(5th cranial nerve from face & jaw)
Gracile and cuneate nuclei
(lower & upper body sensory)
Vestibular nuclei
(equilibrium & reflexive orientation)
Ventral thalamic nuclei
(sensory processing & relay from brainstem & cerebellum to cortex)
Basal ganglia
(subcortical motor control)

Table 2: Areas of Dysmaturation in Brains of Monkeys Following Long-term Survival
Brainstem: periaqueductal gray oculomotor nuclei Inferior olives

Cerebellum:
vermis
Subcortical sites: mammillary bodies hippocampus amygdala
Cerebral Cortex:
frontal and parietal lobes corpus callosum
(left/right hemisphere connection) ventricular enlargement

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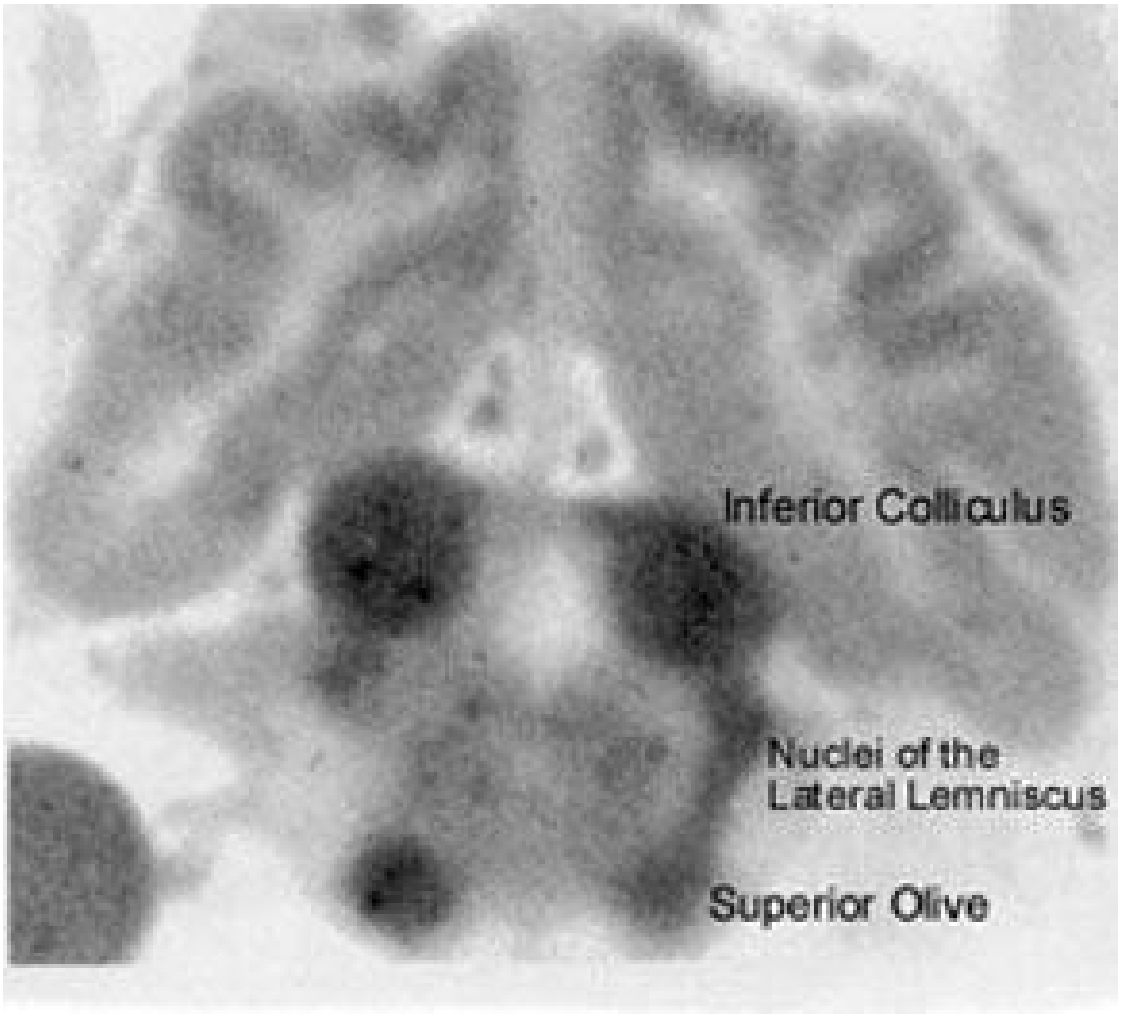

Figure 1: Blood Flow in the Brain
Autoradiogram of the brain of a cat 60 seconds after injection of a radioactive tracer shows the greatest perfusion (thus greatest blood flow) in nuclei of the brainstem auditory pathway. This technique has revealed that the highest blood flow in the brain is to structures in the auditory pathway in several other mammalian species, including monkeys. From Kety, 1962, with permission from Columbia University Press. Note: Labels for components of the auditory pathway, superior olive, nuclei of the lateral lemniscus, and inferior colliculus, were added for reference.
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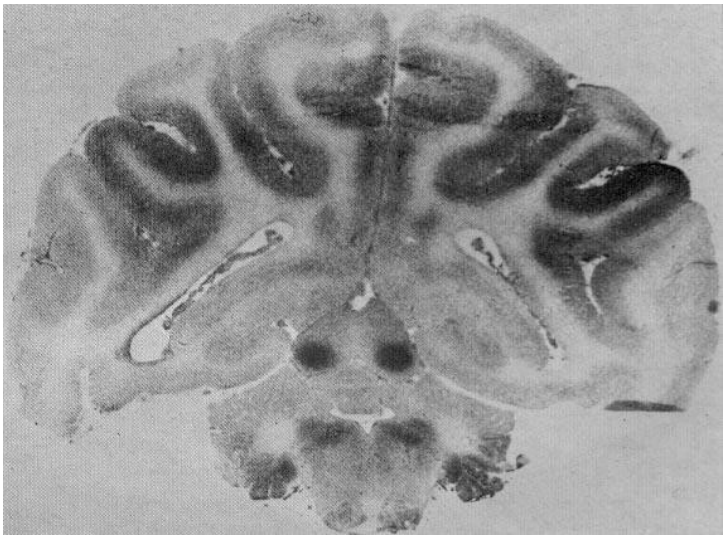

Figure 2. Autoradiograph of coronal brain section of normal newborn monkey. Just prior to being killed, this animal sustained an intravenous infusion of 14C-Iabeled antipyrine. Staining density relates to volume flow of blood per unit of tissue per unit of time. The central nuclei of the inferior colliculi stand out due to their high-volume blood flow< (Courtesy C. Kennedy and L. Sokoloff, Laboratory of Cerebral Metabolism, National Institute of Mental Health.)

Figure 2: Figure 7 from Myers (1972), page 254
Autoradiograph showing blood flow in the brain of a newborn monkey. The greatest blood flow is to the inferior colliculi in the midbrain. High blood flow is also seen in other subcortical structures and inner sulci of the cerebral cortex. From Myers (1972) with permission from the American Medical Association.
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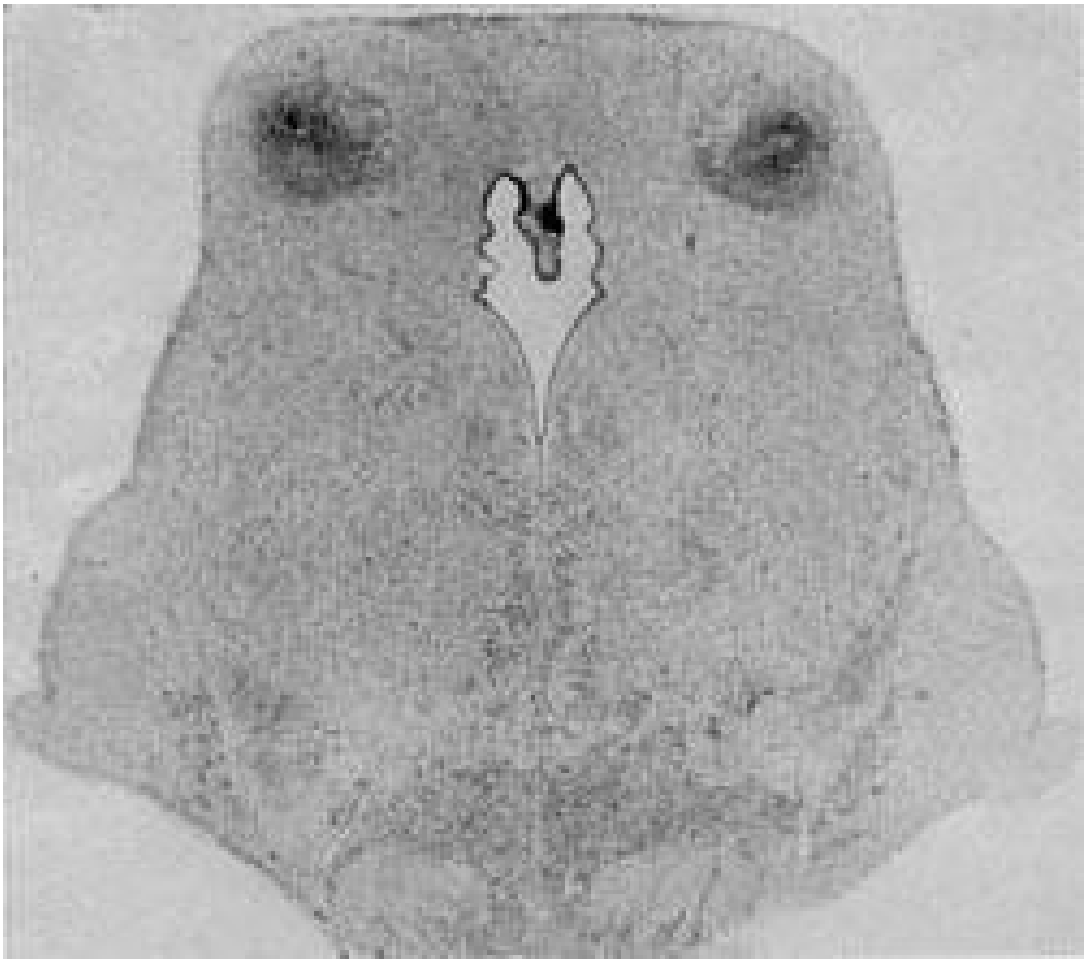

Figure 3: Damage of the inferior colliculi: Result of subjecting a newborn monkey to 12 minutes of total asphyxia. (from Myers 1972)

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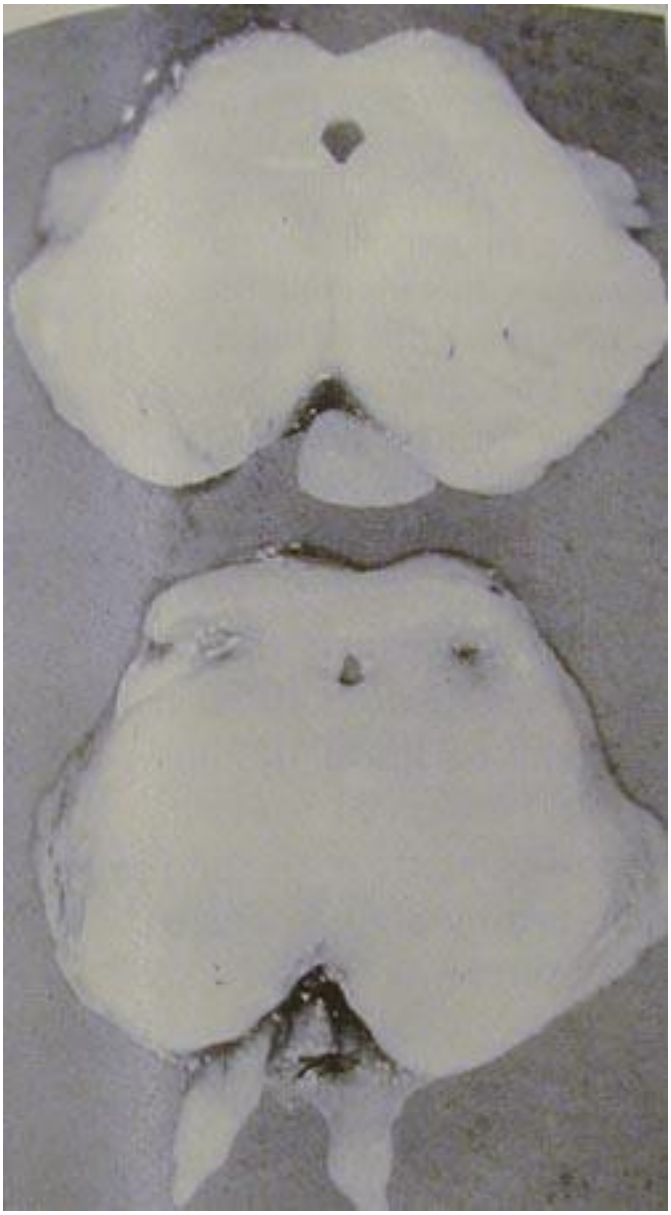

Figure 4: Brain damage in human infants also involves the inferior colliculi (bottom) following asphyxia (from Leech & Alvord 1977, with permission from the American Medical Association).

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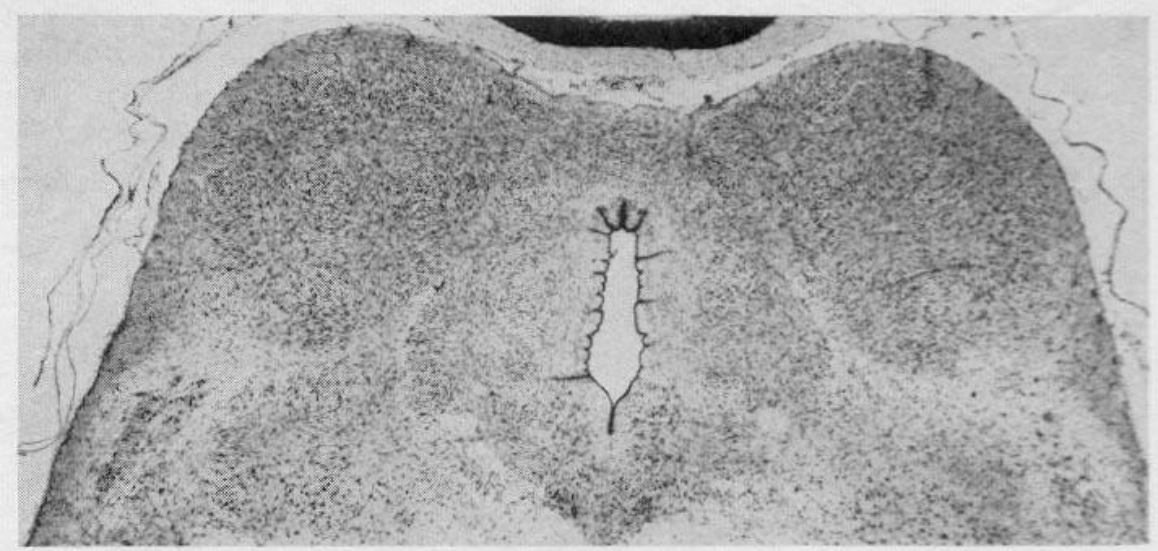
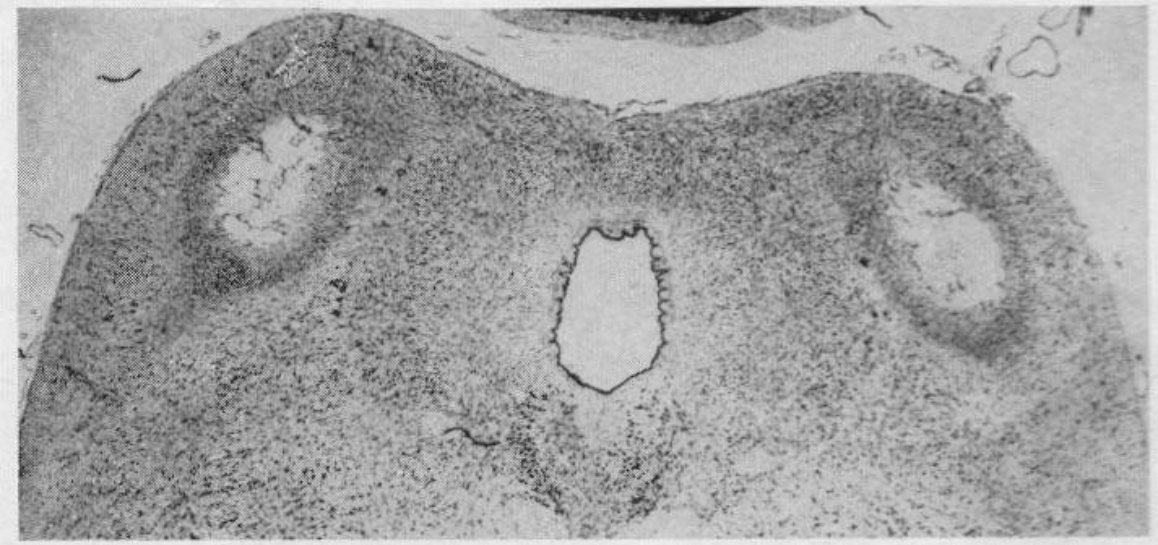


NORMAL COLLICULUS consists of densely packed nerve cells that relay nerve impulses related to hearing originating in the structures of the ear to the higher brain centers.



DAMAGED COLLICULUS from a monkey that was asphyxiated during birth nearly five years previously is pitted by cavities (*left and right*) left by cells that disintegrated.

Figure 5: Pictures from the article by William F. Windle in the October 1969 issue of the Scientific American.

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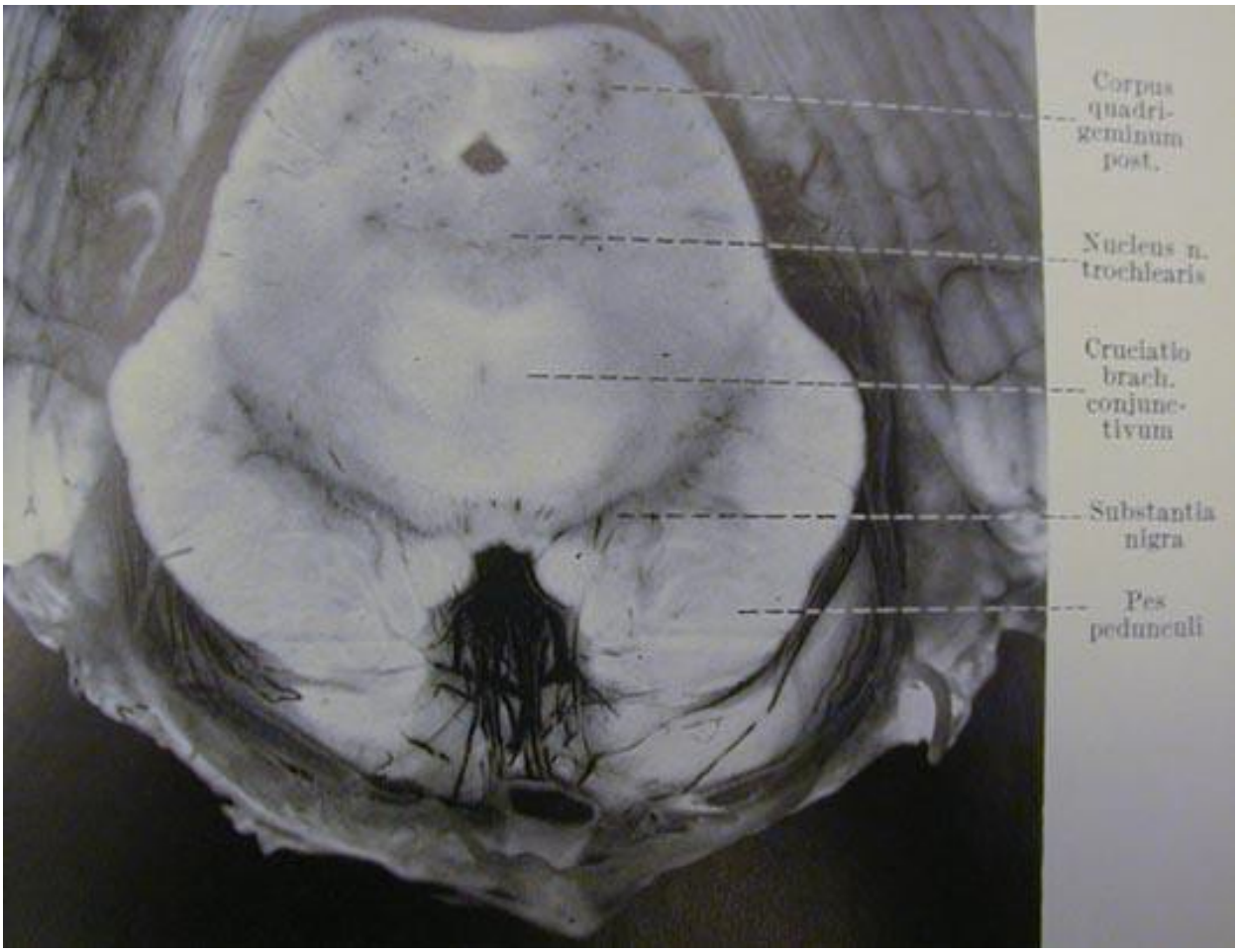

Figure 6: Wernicke's encephalopathy, caused by chronic alcohol intoxication or deficiency of thiamine (vitamin B1) deficiency, is characterized by small flea-bite size hemorrhages in the inferior colliculi (here labeled corpus quadrigeminum posterior) from dilated blood vessels that burst. From Kant (1933, with permission from Springer-Verlag).

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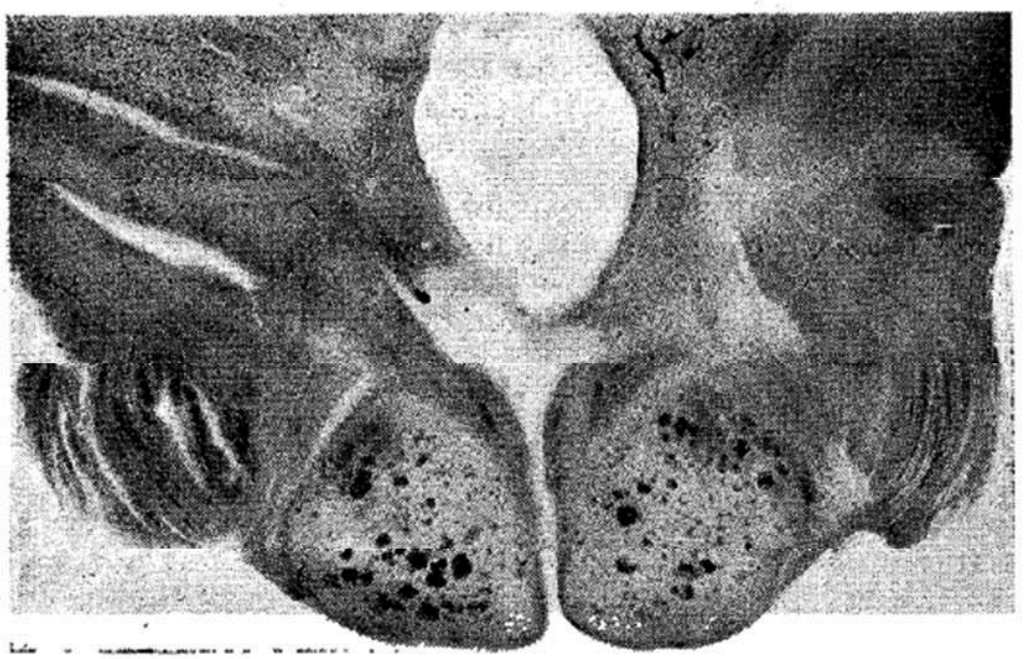

Fig. 1—Frozen section through mammillary bodies showing dilated vessels and hæmorrhages, and a smaller lesion in the wall of the 3rd ventricle. (Lepehne Pickworth, $\times 4^1/_2$.)

Figure 7: Hemorrhagic damage of the mammillary bodies in Wernicke's encephalopathy. From DeWardener and Lennox (1947).

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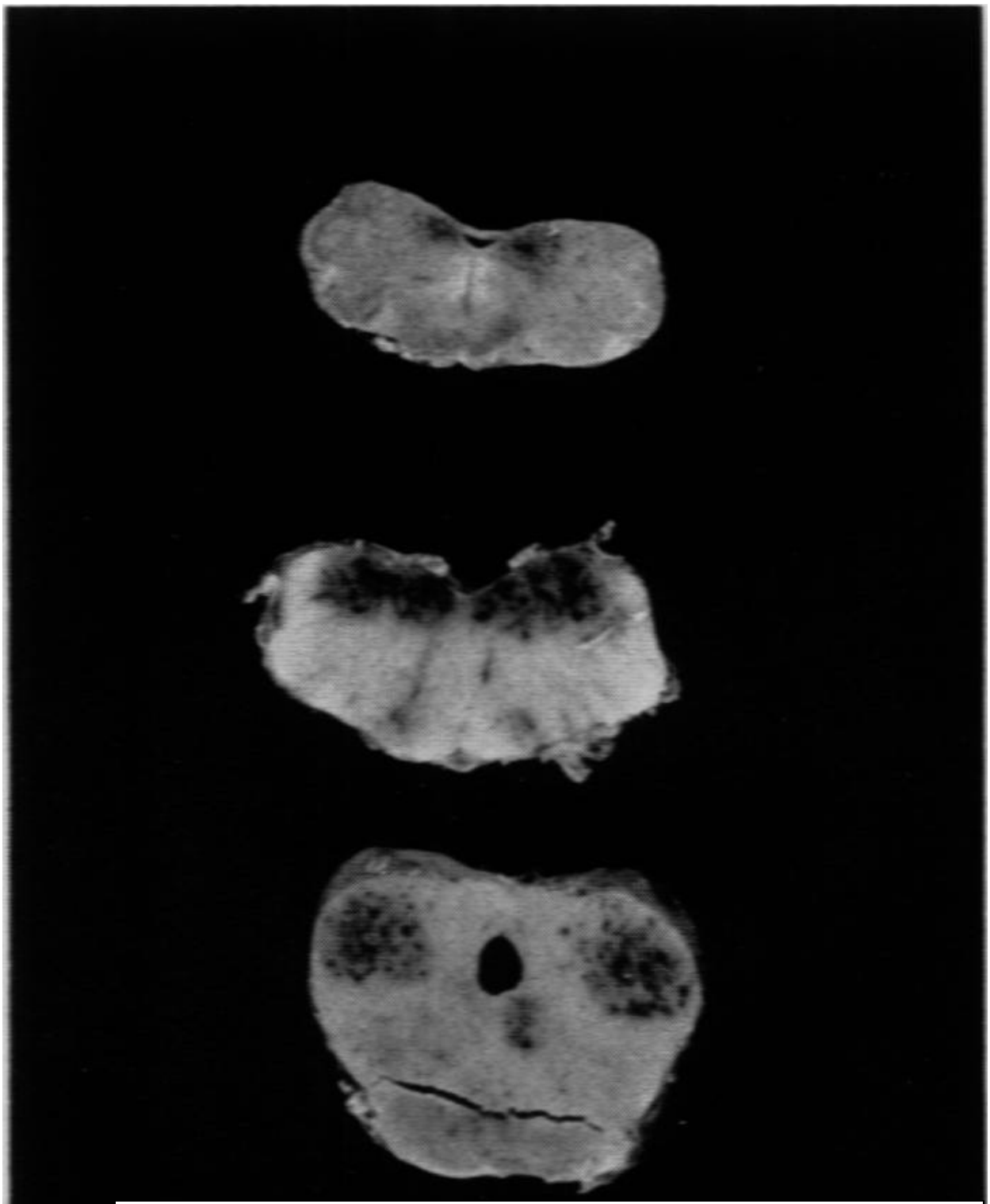

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Figure 1 from Evans et al. (1941) shows the bilaterally symmetric hemorrhagic lesions found in foxes suffering from thiamine deficiency: “The quadrigeminal plate is frequently involved and in all such cases lesions are present in the inferior colliculi.” [p85], as can be seen in the bottom slice shown here.
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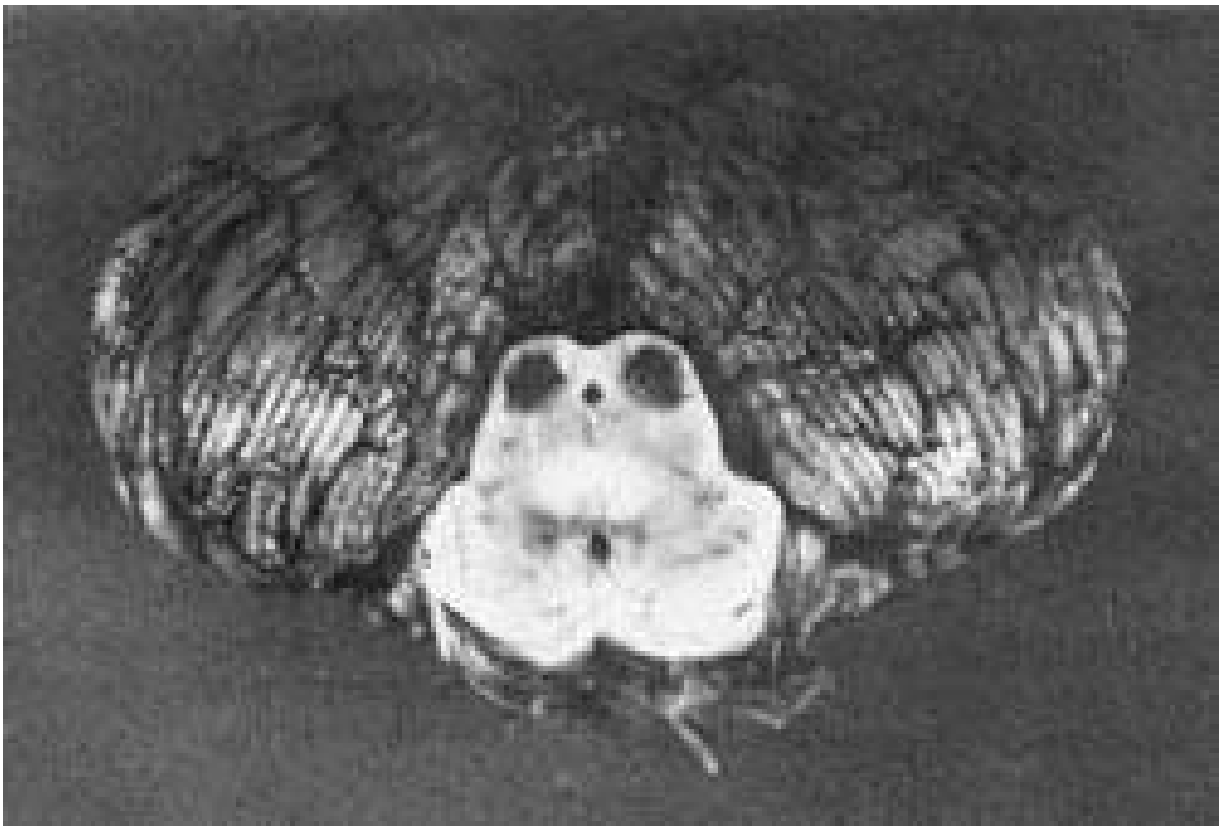

Figure 8: Hemorrhagic damage of the inferior colliculi in a human patient maintained on prolonged parenteral feeding lacking vitamin B1 (from Vortmeyer et al. 1992).

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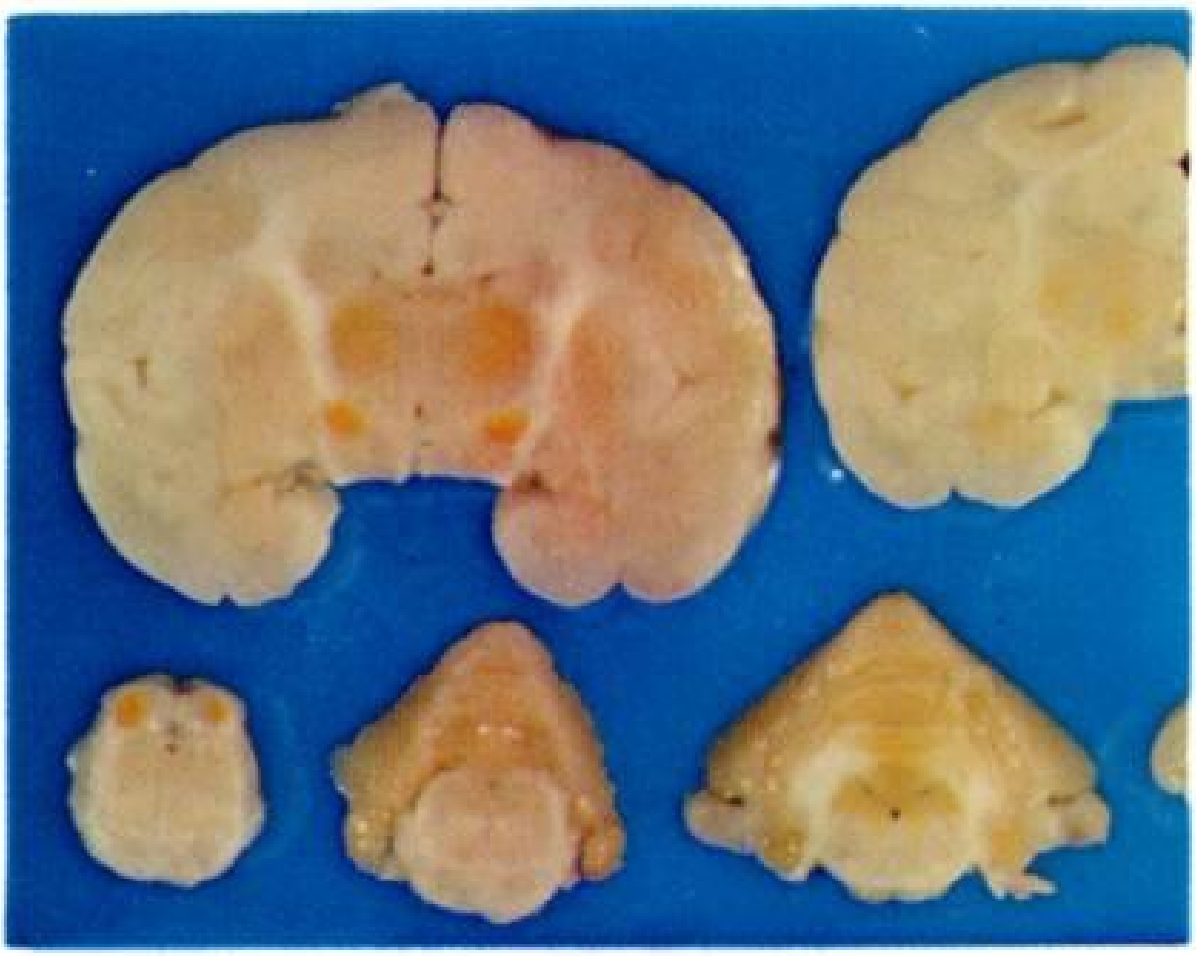

Figure 9: Picture from the the paper by Lucey et al. (1964). Note that bilirubin staining is not uniform throughout the brain. Blood flow and metabolism are not uniform throughout the brain. Brainstem nuclei of high metabolic rate are most susceptible to damage when a sudden catastrophic lapse in aerobic metabolism occurs. The inferior colliculi in the midbrain auditory pathway (lower left) are intensely stained, as are the mammillary bodies and basal ganglia (upper left).

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Note: Personally Identifiable Information (PII) has been redacted in this document

Jean Public

February 3, 2010

Subject: Re: 2010 IACC Strategic Plan Released

THIS AGENCY IS TRYING TO MAKE THIS INTO AN ETERNAL GOLD MINE FOR BIG PHARMA AND BIG MEDICINE. THIS PLAN CONCENTRATES ON AFTER YOU GET IT. IT SHOULD BE CONCENTRATING ON WHY ALL OF A SUDDEN KIDS ARE GETTING IT.YOUR FOCUS IS ALL WRONG AND I THINK YOU PURPOSELY WANT IT THIS WAY SO THAT KIDS WILL CONTINUE TO GET AUTISM. YOU SHOULD BE FOCUSING ON PREVENTION. YOU SHOULD BE DOING TESTS WITH KIDS TO SEE WHAT PREVENTS IT. I THINK YOU WANT ANOTHER CANCER TYPE GOLDMINE THAT GIVES AND GIVES MONEY FOR PROFITEERS. THE FOCUS IS ABSOLUTELY [offensive language redacted] BACKWARDS.

JEAN PUBLIC [PII redacted]

Note: Personally Identifiable Information (PII) has been redacted in this document

Martha England

February 4, 2010

Subject: The lingering questions on ASD and measles mumps rubella (MMR),

I was referred to you by Dr. Catherine Rice (Centers for Disease Control and Prevention (CDC)) who suggested I might be interested in sharing this with your committee.

The reason I am writing you is because after it has be determined over and again that the MMR does not cause ASD, the question still remains on the lips of every parent: Why do they see the onset of ASD appear right around the time they get the MMR vaccine? This question that remains is still responsible for too many children not being given needed vaccines even today. I think I have figured out the answer to that question, and, hopefully, it will help those parent's mind be at rest, to give them a renewed confidence in the vaccine program, and the CDC. It simply seems to be due to a natural process of hemispheric dominance and suppression that no longer masks the underlying deficit causing ASD that becomes apparent only as one hemisphere becomes more dominant and primarily functioning. While I was reading studies about the brain's natural process of Hemispheric Dominance (Hn1 and suppression in development as a result of some other ongoing research I am doing on ASD, this is what occurred to me that you might also find interesting.

It's no surprise that there are hemispheric differences in persons with ASD as well as deficits in automatic processing, and there are plenty of studies to reference that. While doing some reading of studies, I happened to notice many typical right hemisphere (RH) characteristics matched those of many ASD deficit areas, and many typical left hemisphere (LH) characteristics matched those of many ASD hyper - ability areas. I then noticed that the age range of that natural process of hemispheric dominance (HD) was practically the exact same age range as seen in early onset ASD, as well as the same age range when the MMR vaccines are given. I do not know if ASD is the result of problems in that hemispheric process or region. What I do strongly think is that the natural HD process and suppression that occurs explains why it is that we actually begin to see early onset ASD symptoms and why they fade in while abilities fade out. I also think it begins to occur only after both hemispheres are no longer working as much together, thereby no longer compensating for each other, once that natural- HD process begins and suppression of the non-dominant hemisphere takes place. It also seems to me that the degree of the HD process and suppression that takes place has a lot to do with the various levels of ability, severity of motor related issues, language issues, literal interpretation issues and the variety of different, yet, same symptoms of ASD. I think this is because the HD process is in itself very individualized and subject to stimuli. I do not think it is the actual cause of ASD itself.

As I have discussed with Dr. Rice, due to the fact that the ASD onset and symptoms are present in the same exact manner for those with ASD who have never even had the MMR vaccines, I do not feel that the MMR in any way interrupts or interferes with that natural HD process or it's suppression, therefore, is not responsible for the early onset symptoms of ASD that emerge at the same age range they are getting the MMR vaccine. It excludes the MMR as cause of early onset ASD entirely, which concurs with all current findings. Additionally, I feel that the validity of testing for ASD can be questioned up until the

point whereby at natural HD process and suppression finally occurs, along with most allowed hemispheric functionality, as the full scope of the ASD symptoms are probably not apparent until then.

I am not a physician, only the parent of my son, [PII redacted], who has ASD. I do a lot of my own research on ASD and get a lot of exposure due to my job as coach working with this population of individuals. I feel very strongly about this being the actual reason we see the early onset ASD symptoms when we do and I am hopeful that it will put that last lingering question to rest so that families will get their children vaccinated with peace of mind.

Sincerely,
/Martha England/
Martha England
[PII redacted]

Benedetta Stilwell

February 04, 2010

Subject: Information Request on Public Comments

Hi;

I suppose these days there is not many people that thank you for your work. I would like to be able to thank you.

To my sadness that is not possible.

My 23 year old son damaged by a stroke after receiving his third diphtheria, pertussis and tetanus (DPT) shot in 1987, and which left him having to relearn to walk, not talking until the third grade. ---- well --- he is now trying to find a job - after we struggled through high school and a community college. There is no real help for us and I am just as angry about it as I was 23 years ago.

Socially he knows the type of girls that he wants (one with morals) would not look at him twice, so he chooses no girl friend at all.

His health insurance will soon run out and I will have to pay for his epileptic medicine out of my pocket.

Do you suppose some of you could pass a hat around and donate some of the money I pay in taxes, so I can continue to buy his medicine???

I thought not!

I see that this government agency is like all the others - a long string of fancy words and no substance. I suppose all governments finally get to this point where we have people pushing paper and pretending they have a worthwhile job to do.

It could be a worthwhile job if your agency would actually do their job.

I suppose you have won, my life time and my son's life time is just too short while those of your kind can and will continue to suck the life blood out of us. When we are gone, so goes America and your agency helped toward that end.

Thanks,
Benedetta Stilwell of Mt. Vernon, Kentucky

Note: Personally Identifiable Information (PII) has been redacted in this document

Jason Bourret

February 16, 2010

Subject: Behavior Analysis and the IACC Stretegic Plan

Dear Secretary Sebelius and Dr. Insel,

I am a behavior analyst who serves on the editorial board of the Journal of Applied Behavior Analysis and am writing on behalf of the New England Center for Children (NECC), a non-profit school for individuals diagnosed with autism. I have written previously to encourage the addition of behavior analytic and single-subject design expertise to the IACC and I've included that email again below.

I am currently writing in response to the 2010 IACC strategic plan. We at NECC continue to be encouraged by the progress of the IACC and the responsiveness of the IACC to community input. As teachers and therapists working day-to-day with individuals diagnosed with autism, our primary interests lie within the area of Question 4: Which Treatments and Interventions will Help? The need that we have and that we see for those working in other schools is for effective teaching and behavioral treatment methods. Given the immediate need for further sound research on teaching and treatment methodology, we have some concerns about the short and long term objectives listed under Question 4.

Our primary concern is the exclusive focus on randomized control trials (RCT). These are certainly valuable studies that can speak to the overall likelihood of a treatment being effective, or differentially effective, given any selected member of the tested population. We support the allocation of resources to see them conducted but they also have their limitations and of them were discussed following Dr. Charman's presentation to the IACC. RCTs tend to be relatively cumbersome and time consuming. It is difficult to test the sort of nuanced and individualized interventions that are most successful in teaching and treating behavior problems in people diagnosed with autism. Although general effectiveness can be shown, it can be difficult to tease out of date from an RCT the particular necessary and sufficient conditions responsible for treatments being effective in some cases and ineffective in others. There are other research designs that are as experimentally rigorous, and sometimes more so, than RCTs (e.g., within-subject experimental designs involving multiple replications showing the conditions under which effects are and are not found within single experiments) that can be used in both basic and applied research to more rapidly identify novel effective interventions and the particular conditions under which interventions can be expected to be successful. We are concerned that the focus on RCTs to the exclusion of other forms of experimentation may slow the further development of effective teaching and behavioral treatment methods.

Sincerely,

Jason Bourret

Board Certified Behavior Analyst – Doctorate (BCBA-D) Assistant Director of Organizational Behavior Management, New England Center for Children
[PII redacted]

Note: Personally Identifiable Information (PII) has been redacted in this document

Jean Public

February 22, 2010

Subject: REGARDING: 2010 IACC Strategic Plan Released

I WANT TO CALL TO YOUR ATTENTION THAT THE PLAN FUNDS 12 SEPARATE GOVT AGENCIES. THESE AGENCIES DO NOT TALK TO ONE ANOTHER OR SHARE ANYTHING. THEY ALL SEEK MORE AND MORE TAX DOLLARS FOR FUNDING. THERE SHOULD BE ONE AGENCY STUDYING AUTISM. ONE AGENCY INVESTIGATING ALL THE POSSIBLE CAUSES. AND FRANKLY DOES THAT ONE AGENCY HAVE THE [offensive language redacted] TO LAY THE PROBLEM AT THE FEET OF BIG PHARMA VACCINE [offensive language redacted]. I DOUBT IT WITH ALL THE BIG MONEY THAT BIG PHARMA VACCINES [offensive language redacted] SPEND IN LOBBYING WASHINGTON DC - BOTH POLITICAL PARTIES. IT IS CLEAR THAT TAX DOLLARS ARE BEING WASTED HERE. THIS IS MY COMMENT FOR THE PUBLIC RECORD.

JEAN PUBLIC [PII redacted]

Note: Personally Identifiable Information (PII) has been redacted in this document

Martha England

March 18, 2010

Subject: ASD regression To Whom This May Concern,

I am very pleased to read that your new Strategic Plan 2010 will be having a focus on ASD regression. There is regression and then there is "perceived" regression. When my son, [PII redacted], who has ASD, was in grade school he learned how to print words on a page. I suddenly found at about the age of 7 that when he began to learn cursive writing that he was getting the same spelling words wrong. He usually got 100 percent on spelling tests so I looked further. What I discovered was that he wasn't recognizing the print word as the same as the cursive word. They were each exclusive to him.

I devised a spelling sheet for the teachers to print each word in one column, put it in cursive in the next column and then the meaning in a next column. This eventually trained his system to recognize words associated in either manner. Typically, it could have looked like it was regression, as that is the exact age group that regression is mostly seen. At that time, a study on ASD regression was being done somewhere, I don't recall, where I made the statement of a definite distinction needed to be identified between what was real regression and what was simply ASD inability to make transitions or recognize for associations. An ASD learning hurdle is a whole lot different than actual regression of a disorder. I'm afraid there may have been too many people given meds on that assumption for ASD regression when it may have been something else quite simple.

Sincerely, Martha England

Benedetta Stilwell

April 08, 2010

Subject: research sedimentation (sed) rates of mothers

Hi:

How hard would it be to take the C reactive protein levels, and the SED rates of a group of women, if they have an autistic child check and see if these are higher than most that had normal babies? That is of course if there really is an interest in changing the motto of autism is a mystery! Prove it.

Benedetta Stilwell of Mt. Vernon, Kentucky

Eileen Nicole Simon

April 14, 2010

Subject: Regarding: Upcoming Conference Call and Webinar of the IACC Subcommittee for Planning the Annual Strategic Plan Updating Process

I will be on a tour planned many months ago that will prevent my viewing the meetings on April 19 and 30. I will also be at a conference in Europe with my husband on July 16. I am most interested in hearing discussion of public comments and participating in discussion - and hopefully back and forth conversations will be possible.

Attached is a 5-page summary of some papers published in 2009 that I think should be read and discussed by members of the IACC. Also, I have posted some discussion points that I submitted in an earlier email at: http://conradsimon.org/IACCdiscussionPoints.html

Please use the navigation pointers (<< and >>), and the side-bar links to read more. Thanks.

Sincerely,
Eileen Nicole Simon
--
Conrad Simon Memorial Research Initiative
To seek understanding of brain system impairments in autism.
http://conradsimon.org/

[Attachment]:

2009 papers important for understanding autism Eileen Nicole Simon, PhD, Registered nurse (RN) [PII redacted]

Following are some papers published in 2009 that I feel should be read and discussed by members of the IACC:

The following two papers by Russo et al. are most important because they (a) address the core handicap of children with autism, developmental language disorder, and (b) underlying impairments in the brain are investigated:

Russo N, Nicol T, Trommer B, Zecker S, Kraus N. Brainstem transcription of speech is disrupted in children with autism spectrum disorders. Developmental Science. 2009 Jul;12(4):557-67. Demonstration that speech sounds are distorted at the brainstem level in children with ASD.

Russo N, Zecker S, Trommer B, Chen J, Kraus N. Effects of background noise on cortical encoding of speech in autism spectrum disorders. Journal of Autism and Developmental Disorders. 2009 Aug;39(8):1185-96.

Demonstration that cortical processing of speech sounds is defective in children with ASD.

Goldman S, Wang C, Salgado MW, Greene PE, Kim M, Rapin I. Motor stereotypies in children with autism and other developmental disorders. Developmental Medicine and Child Neurology. 2009 Jan;51(1):30-8.

Observations on repetitive stereotyped movements and their correlation with neurological impairment rather than self-stimulation. Impairment of frontostriatal basal ganglia circuitry and cerebellum is suggested as part of early life disruption of subcortical neuronal networks.

Bilder D, Pinborough-Zimmerman J, Miller J, McMahon W. Prenatal, perinatal, and neonatal factors associated with autism spectrum disorders. *Pediatrics* 2009;123;1293-1300.

Use of birth certificate data for statistics on perinatal risk in children with autism compared with a large number of control subjects. Autism was associated with breech presentation and primary (as opposed to repeat) cesarean section. Because breech presentation is a rationale for cesarean delivery, it was not cited as a primary risk factor. Breech presentation was cited as a shared rather than causal risk factor for autism. Other findings included over-representation of firstborn and advanced maternal age.

Comment: This paper like other epidemiology studies does not even mention how the brain might be affected by breech or cesarean birth. The concern is that the infant will suffer oxygen deprivation. An infant born by cesarean section is less ready to give up placental respiration and begin the transition to lung inflation. This along with other epidemiology reports adds to the evidence that perinatal complications are associated with autism.

May-Benson TA, Koomar JA, Teasdale A. Incidence of pre-, peri-, and post-natal birth and developmental problems of children with sensory processing disorder and children with autism spectrum disorder. Frontiers in Integrative Neuroscience. 2009;3:31 pp1-12.

This is one of very few recent papers to cite the work of William Windle:

“Windle (1969) found that monkeys deprived of oxygen at birth demonstrated poor sensory processing and had damage to auditory and tactile processing areas of the brain.” [page 31]

This was an exploratory retrospective study of 1000 children with sensory processing disorder (SPD) and 467 with autism spectrum disorder (ASD) attending a clinic for occupational therapy. Assisted deliveries (C-section, induced labor, forceps and/or suction) were reported in 43.5 percent of children with ASD, and jaundice was reported in 30.2 percent. Delays in motor milestones (rolling over, sitting, crawling, standing, and walking) were commonly reported in addition to developmental language disorder.

Comment: Finding possible causal factors for autism is an urgent problem at the present time. Retrospective chart reviews are often unfairly discredited, but how do insurance companies collect data? No specific unifying features were found in this study, but the authors suggested that, “a number of subtle pathologic factors may occur in combination, and may be influenced by genetics” [page 2]. Perinatal dangers need to be looked at. Obvious problems like prenatal use of drugs and clamping off umbilical blood flow before the first breath should be stopped now.

See also the recent (2010 article: Zhang X, Lv CC, Tian J, Miao RJ, Xi W, Hertz-Picciotto I, Qi L. Prenatal and Perinatal Risk Factors for Autism in China. Journal of Autism and Developmental Disorders. 2010 Apr 1. [Epub ahead of print] Birth injury needs to be a focus of research as a predisposition for autism.

6. Whitney ER, Kemper TL, Rosene DL, Bauman ML, Blatt GJ. Density of cerebellar basket and stellate cells in autism: evidence for a late developmental loss of Purkinje cells.
Journal of Neuroscience Research. 2009 Aug 1;87(10):2245-54.

Cerebellar neurons in brains from six subjects with autism and four controls were counted. Differences approached but did not meet t-test requirements for statistical significance. Discussion of long-known decreased numbers of Purkinje cells points to loss late in gestation or in the postnatal period. Discussion of one prominent outlier autism case underscores diversity of those afflicted with autism.

Comment: Autism is associated with many diverse medical conditions that must all affect the "final common pathway" in the brain. The cerebellum is part of the final common pathway, perhaps responsible for motor disturbances in some individuals with autism, but probably not responsible for the core handicap of developmental language disorder. This paper adds useful evidence from investigation of brain tissue, which must continue to be done.

More articles relevant to autism published in 2009:

Bhutani VK, Johnson L. Kernicterus in the 21st century: frequently asked questions. Journal of Perinatology. 2009 Feb;29 Suppl 1:S20-4.

Brun CC, Nicolson R, Leporé N, Chou YY, Vidal CN, Devito TJ, Drost DJ, Williamson PC, Rajakumar N, Toga AW, Thompson PM. Mapping brain abnormalities in boys with autism. Human Brain Mapping. 2009 Jun 24. [Epub ahead of print]

Buhimschi CS, Weiner CP. Medications in pregnancy and lactation: part 1. Teratology.
Obstetrics and Gynecology. 2009 Jan;113(1):166-88. Erratum in: Obstet Gynecol. 2009 Jun;113(6):1377.

Buhimschi CS, Weiner CP. Medications in pregnancy and lactation: Part 2. Drugs with minimal or unknown human teratogenic effect. Obstetrics and Gynecology. 2009 Feb;113(2 Pt 1):417-32.

Coleman MR, Davis MH, Rodd JM, Robson T, Ali A, Owen AM, Pickard JD. Towards the routine use of brain imaging to aid the clinical diagnosis of disorders of consciousness. Brain.
2009 Sep;132(Pt 9):2541-52.

Fuentes CT, Mostofsky SH, Bastian AJ. Children with autism show specific handwriting impairments.Neurology. 2009 Nov 10;73(19):1532-7.

Hardan AY, Libove RA, Keshavan MS, Melhem NM, Minshew NJ. A Preliminary Longitudinal Magnetic Resonance Imaging Study of Brain Volume and Cortical Thickness in Autism. Biological Psychiatry. 2009 Jun 10.

Hertz-Picciotto I, Delwiche L. The rise in autism and the role of age at diagnosis. Epidemiology. 2009 Jan;20(1):84-90.

Ito T. Children's toxicology from bench to bed--Liver injury (1): Drug-induced metabolic disturbance--toxicity of 5-FU for pyrimidine metabolic disorders and pivalic acid for carnitine metabolism. Journal of Toxicology Science. 2009;34 Suppl 2:SP217-22.

Kuhara T, Ohse M, Inoue Y, Shinka T. Five cases of beta-ureidopropionase deficiency detected by GC/MS analysis of urine metabolome. Journal of Mass Spectrometry. 2009 Feb;44(2):214-21.

Li H, Steyger PS. Synergistic ototoxicity due to noise exposure and aminoglycoside antibiotics. Noise Health. 2009 Jan-Mar;11(42):26-32.

Mercer J, Bewley S. Could early cord clamping harm neonatal stabilization? Lancet. 2009 Aug 1;374(9687):377-8.

Moncrieff J. A critique of the dopamine hypothesis of schizophrenia and psychosis. Harvard Review of Psychiatry. 2009;17(3):214-25.

Mosconi MW, Cody-Hazlett H, Poe MD, Gerig G, Gimpel-Smith R, Piven J. Longitudinal study of amygdala volume and joint attention in 2- to 4-year-old children with autism.
Archives of General Psychiatry. 2009 May;66(5):509-16.

Odd DE, Lewis G, Whitelaw A, Gunnell D. Resuscitation at birth and cognition at 8 years of age: a cohort study. Lancet. 2009 May 9;373(9675):1615-22.

Reiman M, Parkkola R, Johansson R, Jääskeläinen SK, Kujari H, Lehtonen L, Haataja L, Lapinleimu H; PIPARI Study Group. Diffusion tensor imaging of the inferior colliculus and brainstem auditory-evoked potentials in preterm infants. Pediatric Radiology. 2009 Aug;39(8):804-9.

Smith HS. Opioid metabolism. Mayo Clinic Proceedings. 2009 Jul;84(7):613-24.

Stevenson DK. Commentary on the bilirubin supplement. J Perinatol. 2009 Feb;29 Suppl 1:S2-3. Sullivan EV, Pfefferbaum A. Neuroimaging of the Wernicke-Korsakoff syndrome.
Alcohol and Alcoholism. 2009 Mar-Apr;44(2):155-65.

Toal F, Bloemen OJ, Deeley Q, Tunstall N, Daly EM, Page L, Brammer MJ, Murphy KC, Murphy DG. Psychosis and autism: magnetic resonance imaging study of brain anatomy.
British Journal of Psychiatry. 2009 May;194(5):418-25.

Johanna Goldberg

April 19, 2010

Subject: New Cerebrum Article: "Solving the Puzzle of Autism"

I thought you might be interested in a new article, “Solving the Puzzle of Autism,” from *Cerebrum*, the Dana Foundation’s online magazine. It focuses on the genetic, neuronal, and behavioral elements at play in autism, and why these elements may be even more complex than those involved in cancer. The article is available at http://www.dana.org/news/cerebrum/detail.aspx?id=26886. Please share it with your members and colleagues.

In the article, Dr. Alan Packer, associate director for research at the Simons Foundation Autism Research Initiative, describes the difficulties of developing successful remedies for arguably less complex diseases than autism. Due to the scope and complexity of autism as a neuropsychiatric disorder, he believes the development of effective treatments will require a long-term, multidisciplinary approach.

Best,

Johanna

Eileen Nicole Simon

April 23, 2014

Attached is an 8-page pdf file I want to submit as written comments to members of the IACC. Thanks for your helpfulness.

Sincerely,
Eileen Simon
--
Conrad Simon Memorial Research Initiative
To seek understanding of brain system impairments in autism.
http://conradsimon.org/

Eileen Nicole Simon's attachment can be viewed here:
Attachment (PDF – 461 KB)

Caroline Rodgers

April 26, 2010

Subject: corrections to January IACC advance copy

Good morning.

First of all, I want to thank Dr. Insel and the rest of you serving on the Interagency Autism Coordinating Committee (IACC) for allowing me this opportunity to share my thoughts about the 2010 Strategic Plan. You have undertaken a huge, multifaceted task that has only become more difficult since you began. I hope that my comments today will help you in your endeavors.

In the spirit of full disclosure, I am the author of the article, "Questions about Prenatal Ultrasound and the Alarming Increase in Autism," published in Midwifery Today in 2006 and reprinted in Pathways to Family Wellness last summer. I have no financial, professional or personal interest in either prenatal ultrasound or autism, although like you, I am deeply concerned about the autism crisis. Driven by this concern, I have continued my research, which has given me a unique perspective in interpreting new autism findings.

I know that the IACC has put many long, tedious hours into updating the Strategic Plan and that you hope to approve it today. The line-by-line edits you made in Section III, "What Caused This to Happen and Can This Be Prevented," were made on November 10th. Since then, like movies vying for Oscar consideration that open the last month of the year, important and intriguing studies have been published that raise new questions about what is causing autism. There is even the possibility that the relentless increase in autism could be reversed in the time it takes to yank an electrical plug out of its socket. At the end of this talk, I have specific, line-by-line suggestions for making changes in Section III. Due to time constraints, I will be unable to present these proposed changes orally, but they are available to the committee in the document I submitted in advance and will be available to all others as part of the public record.

Alabama's intriguing Hispanic anomaly
On December 18th – a little more than a month after the IACC revised Section III – the Centers for Disease Control and Prevention (CDC) published shocking figures that showed a 57% increase in autism (1). I'm sure your hearts sank when you saw those numbers – I know mine did. But a closer look at the facts revealed a puzzling anomaly: At the same time the overall autism rate charged ahead 57 percent, it DECREASED 68 percent among Alabama Hispanics. Hey – that's the direction we want the whole nation to go! I wondered what could possibly have changed in Alabama's public health policy that could have caused such a great about-face among the Hispanic population. Was there a special outreach program or an increase in public assistance? Imagine my surprise when I discovered a 2002 CDC report revealed that during the time the mothers of the autistic children were pregnant, Alabama, was one of three states to cut Medicaid for prenatal care (2).

Could prenatal care cause autism? That's crazy talk!

Maybe not: Alabama and Florida – two of the three states that cut public assistance for prenatal care from 1993 to 2002 – had the lowest autism rates of the 11 states that participated in the CDC's

surveillance program. Did this correlation between lower autism rates and lack of prenatal care hold up throughout the study?

There were significant ethnic differences in the autism rates reported in last month's CDC report. White women had a higher rate of children diagnosed with autism than either black or Hispanic women. Per 10,000 women, the autism rate for children of non-Hispanic white women was 101, versus 76 for black women and only 61 for Hispanic women. The numbers provide substantial and irrefutable ethnic differences in autism rates, but was there a correlation with prenatal care?

The answer is yes – although I had to delve into another CDC multistate surveillance report to find it. The report, regarding the timing and entry into prenatal care from 1989-1997, showed that more than twice the percentage of black and Hispanic mothers lacked first-trimester prenatal care than did non-Hispanic white women. By 1997, although the overall percentage of women who received first-trimester prenatal care increased, the ethnic differences remained (3).

Taken together, these three CDC reports tell a disturbing story: as more women across all ethnic groups received first-trimester prenatal care, the autism rate among their children increased, with greater increases among the groups that had more first-trimester prenatal care. This seems counter-intuitive: Shouldn't first-trimester prenatal care result in healthier children? While these studies do not prove causation or even a correlation between prenatal ultrasound and autism, they raise the question:

What aspect of prenatal care may place babies at greater risk of autism?

Poked, prodded, measured – and scanned

While the autism rate has increased dramatically in the last three decades, very little has changed regarding prenatal care. The pregnant woman is weighed and her vital signs are taken. The height of her expanding belly is measured and her ankles are checked for swelling. She will be asked to give blood and urine samples to check for diabetes and preeclampsia, prescribed prenatal vitamins with folic acid to protect against spina bifida, advised about diet and warned regarding the dangers of smoking, alcohol and recreational drugs.

What HAS changed in prenatal care in the last few decades has been the addition of ultrasound to monitor pregnancies. Like many interventions that were initially used only in high-risk groups, reliance on prenatal ultrasound rapidly became standard practice for most pregnancies. Prenatal ultrasound has also undergone rapid and extensive changes in terms of the technology, different applications, the number of scans per pregnancy, as well as the gestational window of exposure, which has extended to all three trimesters. Therefore, although every aspect of prenatal care should be carefully reconsidered, prenatal ultrasound deserves especially close scrutiny.

Say it ain't so: Educated moms are at higher risk of having children diagnosed with autism
A University of California (UC) Davis study published this month found 10 autism clusters in California and concluded that highly educated white parents were more likely to have children diagnosed with autism. In six of the clusters, couples in which one parent graduated from college had four times the rate of having an autistic child than parents in the same area that did not finish high school (4).

This seems counter-intuitive: Don't highly educated white mothers hold all of the cards? They enjoy

educational opportunities, have their pick of the best jobs and receive the finest medical care available -- Why wouldn't their children be the healthiest? Could the best obstetrical care available have a down side? What if the use of prenatal ultrasound, considered essential to monitoring pregnancies, sometimes backfires?

Highly educated white women are more likely to undergo prenatal ultrasound because they are more likely to . . .

1.) have health insurance

2.) start prenatal care in the first trimester

3.) independently research prenatal care and do everything they can to assure healthy pregnancies

4.) be able to afford and even demand diagnostic imaging they believe will help ensure a healthy baby

5.) defer having children until they are older, due to education or career pursuits, ultimately placing themselves in the —high-risk category of older moms who undergo amniocentesis or chorionic villus sampling — both of which are guided by ultrasound

6.) have disposable income that can pay for —extras, such as the keepsake prenatal ultrasound portraits and Digital Versatile Discs (DVDs) that are available at many malls

7.) Finally, if they have fertility issues, the —older, white and highly educated parents are more likely to be able to afford and undergo assisted reproduction, which involves additional ultrasound scans

Canadian moms have 55 percent more scans per pregnancy while autism rate rises 50 percent
It is difficult to find hard data on prenatal ultrasound use. In the multibillion-dollar ultrasound industry, this information has value and is not freely available, so a Canadian study published this month helps provide an idea of industry trends.

The study showed that for the 10-year period ending in 2006, there was a 55 percent increase in the number of ultrasound scans per pregnancy among Ontario women, with the biggest increase in additional ultrasound scans in routine pregnancies, rather than in high-risk pregnancies (5).

Although these figures apply only to Ontario, principal investigator Dr. John You, noting that "a 55 percent increase is pretty substantial," told the Canwest News Service: "I wouldn't be surprised if we saw similar findings across the country."

Meanwhile, Canada's autism rate has increased 50 percent, from 1 in 250 live births to 1 in 165, according to the Autism Society of Canada (6).

While these two independent statistics regarding ultrasound exposure and an increase in autism do not provide causation or even correlation, they are red flags that deserve further investigation.

New facts raise new questions

The results of the three studies I have discussed today – one showing that ethnic groups with the least access to prenatal care had fewer autistic children, a second showing that women who had educational, economic and medical advantages had children with a much higher autism rate and the third indicating that the number of ultrasound scans per pregnancy has increased dramatically – suggest that we start asking new questions.

Do some people have genetic predispositions that make them more susceptible to ultrasound-induced damage? Early studies showed that ultrasound can damage mitochondria – could ultrasound be causing non-inherited mitochondrial disorders that lead to autism? Ultrasound heats tissue – could this damage heat-shock proteins, hampering their ability to protect newly vaccinated children experiencing prolonged, high fevers? Could the thermal effects of ultrasound be causing changes in gene expression that result in autism?

We need to ask whether fetal brains sometimes are being harmed by the very imaging technology intended to help ensure healthy pregnancies. It is a horrifying possibility. The good news is, if prenatal ultrasound is causing autism, strict rules regulating its use could cause a swift turnaround in the autism rate. Picture that!

Specifically, I am asking the IACC to consider making the following changes to Section III of the Strategic Plan.

1.) On Page 1, "What caused this to happen and can this be prevented?" I suggest adding the bullet: What aspect of prenatal care may be increasing the risk of ASD?

2.) On Page 5, line 17 after the sentence ending in the word "risk." I recommend adding: "New studies regarding ethnic differences in autism rates, combined with previous studies regarding entry into prenatal care, indicate that some aspect of current prenatal care may contribute to causing autism."

3.) On Page 5, line 21 after the sentence ending in the acronym "(EPA) (Environmental Protection Agency)." I recommend adding: "Wherever possible in the previously sited studies, efforts should be made to record data regarding ultrasound exposure including the output power, type and length of examination, as well as information regarding optional, keepsake ultrasound sessions."

4.) On Page 9, Line 18, I recommend a new paragraph, as follows: "Since prenatal ultrasound exposure may prove to be a factor in causing autism, we need to provide all ultrasound operators with a standard format for recording the output power, type and length of examination, along with any other factors recommended by experts who specialize in fetal ultrasound safety. This format must be required, so that accurate data in a standard format are available for future analysis."

5.) On Page 10, remove the word "ultrasound" from the seventh bullet listing potential environmental factors. Instead, add the following bullet: "Changes in prenatal care, specifically changes in the application, technology (including but not limited to increases in potential acoustic output, harmonic imaging, Doppler imaging, both spectral and color, 3-dimensional imaging and ultrasound contrast agents) and gestational window of exposure for prenatal ultrasound.

If the IACC agrees to incorporate the above suggestions, then additions will also need to be made in both the short-term and long-term objectives sections.

In closing, I want to extend my deepest gratitude to the IACC for the time and attention you have given to these ideas. Your task is not an easy one and it is far from done. I am sure you appreciate that if prenatal ultrasound is causing autism, then every delay of even one day will cause unnecessary, untold heartache for families across the nation. I hope that you will find the information and insights I shared with you today useful in updating the 2010 Strategic Plan.
Thank you.

Caroline Rodgers

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