year, some 40,000 are born with a “critical” form of CHD that will require intensive surgical intervention within the first year of life. As recently as the 1980s, children born with some forms of critical CHD did not survive; palliation was their only option. This has changed dramatically over the past 30-40 years. Driven by momentous breakthroughs in medical science and technology, approximately 80-95% of children born with CHD today will reach adulthood.

But increased survival is only a part of the CHD story. Indeed, like extreme prematurity, leukemia, and many other previously fatal medical conditions with which neuropsychologists are familiar, increases in longevity among CHD survivors have come with increasing recognition of the many challenging transitions and cumulative medical, neurobehavioral, and psychosocial burdens inherent to “living with CHD.”

CHD begins to alter expected brain development in utero with evidence of structural, volumetric, and metabolic differences documented as early as the second or third prenatal trimester. Brain dysmaturation, in turn, increases one’s risk for further acquired brain injury and gives rise to a range of neurobehavioral deficits and psychosocial difficulties that consistently rank among the most salient threats to quality of life among children, adolescents, and adults with CHD.

More recently, as survival into adulthood has become increasingly common for individuals with CHD, we have also begun to more fully appreciate the cascading impact and cumulative neuropsychological burden of CHD across the lifespan, which impact a range of long-term outcomes such as educational and occupational attainment, living independently, and risk for dementia.

In short, CHD can no longer reasonably be considered a child or pediatric condition, but rather a lifespan condition with the potential to adversely impact neurobehavioral and psychosocial outcomes in different ways and at different times across infancy, childhood, adolescence, and adulthood.

Over a series of talks presented by a panel of recognized neuropsychologists and experts in CHD, this symposium aims to review the neuropsychology of CHD across the lifespan and to present an integrative lifespan developmental neuropsychological model of CHD that eschews prevailing “child” vs. “adult” distinctions. Each presentation will address a salient developmental epoch (prenatal-early childhood, school-age/adolescence, and adulthood/geriatric timeframes) and will include a comprehensive review of the extant literature pertaining to relevant neuroanatomical and neurodevelopmental/neuropsychological considerations for individuals with CHD during each epoch. Transitions, of which there are myriad for individuals living with CHD (e.g., from acute inpatient care to stepdown unit care; from inpatient to outpatient settings; from early intervention to the school system; from childhood to adolescence; from adolescence to young adulthood; from pediatric to adult CHD care), will feature prominently throughout the symposium, as will recommendations for competent, developmentally-informed clinical neuropsychological management and intervention planning throughout the lifespan.

Upon conclusion of this course, learners will be able to:

1. Describe the mechanisms by which congenital heart disease (CHD) impacts brain development and functioning across the lifespan (from infancy to older adulthood).
2. Discuss neurodevelopmental/neuropsychological sequelae of CHD for children, adolescents, and adults.
3. Explain the role of clinical neuropsychologists in evaluating, supporting, and optimizing the neuropsychological trajectories of individuals with CHD across the lifespan.

Poster Session 01: Medical | Neurological Disorders | Neuropsychiatry | Psychopharmacology

2:45 - 4:00pm
Wednesday, 1st February, 2023
Town & Country Foyer

1 Exploratory Factor Analysis of the Core Neurocognitive Syndrome in Agenesis of the Corpus Callosum

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Objective: A previous review of the syndrome of Agenesis of the Corpus Callosum (ACC) identified three primary deficits: reduced interhemispheric transfer of sensory motor information, slowed cognitive processing, and deficits in complex problem solving (Brown & Paul, 2019). Interaction of these three deficits contributes to a variety of secondary cognitive and psychosocial deficiencies across domains. Recent research has also identified a possible fourth core deficit in ACC: restricted capacity for elaborative thought and creativity (Renteria et al., 2022; Bogen & Bogen 1988). We examined the syndrome of ACC using an exploratory factor analysis of neuropsychological test data (not including data regarding interhemispheric transfer) and hypothesized it would organize into factors of (1) reduced cognitive processing speed, (2) difficulty with complex problem solving, and (3) difficulty with creative tasks.
Participants and Methods: The present study analyzed archival data from individuals with ACC (N=60) acquired from common neuropsychological instruments: D-KEFS, WAIS-III, and WRAT-2. Among the participants, 13 had partial ACC, 1 was unspecified, and the remainder had complete ACC. The participants' ages ranged from 7 to 55 years (M = 21.55, SD = 12.36), with an education level that ranged from 2 to 19 years (M = 11.59, SD = 3.77). All participants had complete data for at least one assessment and all available data was included. Missing values (49.85%) were excluded from analysis. Factor analysis (principal factor solution with promax rotation) was conducted with 33 commonly derived summary (standard) scores. Horn's Parallel Analysis recommended a 4-factor solution, but we elected to generate a 3-factor model that would more closely follow previous literature.
Results: Factor one involved processing speed and was comprised primarily of D-KEFS Color Word Interference Word Reading (1.02) and Color Naming (.78), D-KEFS Trail Making Test Visual Scanning (.86) and Number Sequencing (.74), and WAIS-III Processing Speed Index (.68). The second factor included several problem solving measures [e.g. D-KEFS Sorting Test Free Sorting (.90) and Sort Recognition (.90), and WAIS-III Perceptual Organization Index (.72)], as well as several additional measures including WAIS-III Working Memory Index (.84), WRAT-2 Arithmetic (.83), and WAIS-III Verbal Comprehension Index (.80). Finally, the third factor involved several measures requiring mental flexibility and cognitive control [e.g. D-KEFS Twenty Questions Test Achievement Score (.70), D-KEFS Design Fluency Switching Condition (.56), and D-KEFS Trail Making Test Number-Letter Switching Condition (.44)], as well as a measure of single word reading [WRAT-2 Reading (.66)].
Conclusions: The findings support inclusion of slowed cognitive processing speed as a core feature of the neurocognitive syndrome in ACC described by Brown and Paul (2019). The second factor is partially consistent with a deficit in complex problem solving, but is not restricted to that cognitive domain. Likewise, the third factor is largely related to mental flexibility and control (one aspect of creativity), but is not restricted to that domain. Future attempts to model the neurocognitive syndrome of ACC may provide greater clarity by including a wider range of cognitive and psychosocial indices and excluding individuals with comorbid neuropathology.

Categories: Behavioral Neurology/Cerebral Lateralization/Callosal Studies
Keyword 1: corpus callosum
Keyword 2: cognitive functioning
Keyword 3: intellectual disability

2 Musical perception skills in Agenesis of the Corpus Callosum

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Objective: Agenesis of the corpus callosum(AgCC) is a disorder in which the connection between the two brain hemispheres is congenitally absent. Previous research has suggested that the auditory system can be affected in individuals with AgCC (Demopoulos et al., 2015). However, the nature of AgCC's effect on musical perception skills is unclear. This study investigated the impact of AgCC on the music perception skills in high-functioining adults using a brief version of the Profile of

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