CHAPTER 9

Cybernetic Children

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Cybernetics

- from Greek: kybernetes helmsman (kybernan: to steer, govern)

- the science dealing with the comparative study of human control systems such as the brain and nervous system, and complex electronic systems

The subject of this chapter is the science of *cybernetics as it applies to children*. Two themes emerge: (1) new technologies are changing the human brain in ways that make children increasingly vulnerable to, and dependent upon, electronic systems; (2) pharmacological interventions (illustrated by the stimulant therapies given for so-called Attention Deficit and Hyperactivity Disorder) are a destructive response to these developments. The outcome of both forces is the increasing construction of cybernetic (*electro-chemically* controlled) childhoods, and the destruction of children.

The c*onstruction of childhood* is, for wealthy nations, in the context of television, video games, the Internet, text messages, mobile phones and computers. The *destruction of children* in these wealthy nations increasingly occurs through the prescription of psychiatric medications, specifically stimulants.

Today's children

In his book *Open Sky*, Paul Virilio[1] outlines three discrete phases in history. The First Interval, illustrated by the transportation revolution of the 19th century, represents the interval of space where the journey (geographic expanse) was conquered. The Second Interval, signified by the Transmission Revolution, has

resulted in the abolition of time. In other words, television and radio have enabled simultaneous *reception.* In the Third Interval, signified by light speed and fiberoptic transmission (the world of the Internet), there exists only the world of the instantaneous. Virilio argues that technology has changed what it means to be human. He views mankind as evolving from active traveller to passive recipient of electromagnetic signals. On a social and cultural scale, he worries that new technologies have led increasingly to a distorted form of interactivity, whereby people have become so engaged with on-line pseudo-realities that they have lost touch with the realities around them.

Dromospheric Pollution and the Developing Brain

Dromology is the study of speed (from dromos = race, running):

> Alongside air pollution, water pollution, and the like, there exists an unnoticed phenomenon of pollution of the world's dimensions that I propose to call dromospheric.[2]

Evidence that the dromosphere may be changing our children comes from three recent investigations.

Researchers at the University of Washington's department of paediatrics studied the impact of television exposure in early childhood.[3] The study design involved interviews with children (average age seven) in three different survey waves: 1996, 1998, and 2000. The main outcome measure used was the score each child received on a subscale of the Behavioral Problems Index. This score was then correlated with average hours of TV viewing, based upon interviews with the children's mothers that had been recorded in a separate database many years before.

Even after controlling for the influence of prenatal substance use, gestational age, maternal psychopathology, and socioeconomic status, the research found a strong association between early TV exposure and the development of attentional problems by early childhood: 10 percent increased risk of attentional decrements for every hour of television watched at age one or three.[4] The investigators concluded that their findings were consistent with the recommendations of the American Academy of Pediatrics (urging caution in TV exposure for children under two) and with the findings of other researchers whose studies had confirmed links between television, reduced reading ability, and poor attention spans.

A second study, at the University of Rochester,[5] explored the impact of different types of video games upon four tests of selective visual attention. The study compared the performance of experienced video game players with non-players. A separate experiment involved training the non-video game players and re-testing for signs of improvement. In the first test, subjects were exposed to target symbols and distracting flankers (called a flanker compatibility effect). This test was designed to assess the capacity to ignore detractors on a target task. As the performance task increased in difficulty, experienced video game players

were found to have much greater capacity to avoid irrelevant processing (i.e., able to remain on-task longer). In the second test, called an enumeration task, subjects were asked to identify the number of squares (1 to10) that were presented in briefly flashed displays. Video game players remained highly accurate in their ability to track higher numbers of items at the same time (about 30 percent more items than non-video game players.) In the third test, subjects were expected to identify a localized target against a cluttered background. Video game players performed about 50 percent better than non-players on the task, which examined the processing of visual information over space. In the fourth test, subjects were presented with a list of letters flashed quickly one after another in the middle of the screen. On each trial, the subjects knew that all letters would be black except for one letter. Their task required the identification of the one white letter. They were also required to say whether or not the letter X had been presented (it was shown in only 50 percent of the trials.) Findings revealed that the detection of the letter X depended upon how closely it was related to the appearance of the white letter. If X was flashed within several hundred milliseconds of the white letter, it was missed. Action game players outperformed non-video game players on this task, suggesting an increased ability to process visual information over time: increased task switching, decreased 'attentional blink'.

The investigators concluded that action video games (but not non-action games, such as Tetris) improve several elements of selective visual attention. They then performed a fifth experiment which exposed non-video game players to training (action vs. non-action video game, 1 hour per day for 10 days). All participants improved their scores on the video games on which they had trained. Action game training led to greater improvement on tasks of selective visual attention, spatial distribution, and temporal resolution. When interviewed about the possible implications of their research, the investigators conceded that video games probably 'do *not* cultivate the sustained attention needed for tasks such as reading,' (italics added) but they were optimistic that action games might be used for rehabilitating visually impaired patients (e.g., stroke victims) or for training military personnel.[6] The *possibility that action video games might over-stimulate attentional or visual systems in some age groups was not addressed*, (emphasis added).

Researchers in London[7] investigated the effects of video game playing upon dopamine release in the brain. PET (positron emission tomography) scans were given to eight male volunteers during the first 50 minutes (learning phase) of a new video game. The game involved navigating a tank through a battlefield to collect flags and destroy enemy tanks, with higher scores leading to monetary reward. During a second PET scan, subjects stared at a blank screen. Changes in the binding of a radiolabelled ligand (raclopride, a drug which binds selectively to the D2 receptor) were used to infer differences in dopamine release in the striatum of the brain. The experiment revealed significant increases (at least twofold) in extra-cellular dopamine during video game playing, similar to that seen with intravenous injections of amphetamine or methylphenidate. Results

appeared to validate the findings of electrophysiological studies in animals, suggesting a link between dopamine neurotransmission and sensorimotor functions related to reward, aversive conditioning, and stressful stimuli.

Dromospheric Addiction

The possibility that new technologies might feed addictive patterns of behavior has become a topic of concern for several investigators.[8–11] In 1996, a psychologist at the University of Pittsburgh proposed a modification of the DSM (Diagnostic and Statistical Manual of Mental Disorders) criteria for pathological gambling and substance dependence to create a measure of Internet addiction. More recently, she has developed an Internet Addiction Rating Scale which—ironically—is available to the public on-line.[12] Other researchers have conducted studies which demonstrate 'excessive use' of the Internet among individuals with high lifetime rates of so-called psychiatric conditions, including anxiety and eating disorders, alcohol dependence, and manic depression. In one community survey of 169 Internet users, greater use of the Internet was associated with a decline in social involvement and mood.

The call for the inclusion of 'Internet Addiction Disorder' in the psychiatry lexicon has not been unopposed. At least one observer[13] has questioned the premature creation of a new diagnostic entity on the grounds of unclear construct validity. First, it is not at all clear what constitutes 'normal' versus 'excessive' computer use. Second, there are problems associated with the identification of the appropriate object of addiction: is it the computer use itself, or some other object—such as information, companionship, or on-line purchases—which is compulsively desired? While psychiatric researchers express an interest in identifying the biochemical aspects of compulsive computing, there is little discussion about the motivation which lies behind the behavior. To the extent that researchers focus narrowly upon reward systems and dopamine pathways, they will continue to miss the most important aspect of the phenomenon: the *meaning* which Internet connectivity has come to assume for each individual and for society.

The psychopharmacological destruction of children

New technologies—including television, video games, and the Internet—are changing the brain and changing the nature of human relatedness. Small wonder, then, that children have become increasingly prone to shorter attention spans, increased impulsivity, and hyperkinesis—features which are now understood in terms of the conditioning of dopamine pathways in key centers of the brain. To put it briefly, the dromospheric pollution is creating children who are addicted to novel stimuli, multi-tasking, and speed. What is most concerning, however, is the method which industrialized societies have adopted for dealing with the changed nature of childhood. Consider the following Letter to the Editor which appeared in a 1971 issue of *Pediatrics*:

> I receive one or two calls a week about the first or second grade boy who is not sitting still in school, who is disturbing the other children, and whose parent or teacher or guidance counselor feels that my putting him on methylphenidate hydrochloride (Ritalin) or dextroamphetamine (Dexedrine) would be of value.

Recently a mother called me with this typical case history. I explained to her

> … I had detected no neurological abnormalities. I also pointed out that nothing in his prenatal or subsequent course had indicated neurological impairment … Further discussion centered around the fact that his hyperactivity would probably get better when he was around 9 or 10 years old, and it was worth waiting. It was also pointed out that, really, the long range effects of medication to make boys sit still are not well known.
>
> The boy's mother ended the conversation with a most heartwarming statement: 'In other words, Doctor, what you're telling me is that Ritalin wouldn't make my boy any better but would make the school better'.[14]

Such a letter would be unlikely make it past peer review if it were submitted to the same journal today.

Stimulants and adverse events

Despite the precipitous increase in the use of stimulant medications to control childhood conduct, particularly within the United States, scant attention has been paid to the deleterious effects of these drugs. It is typical of the psychiatric literature to refer to stimulant side effects as 'mild, time-limited, and well tolerated'. For some children, this may indeed be true. For other children, however, the drugs have been harmful, even lethal. A closer inspection of both the acute and long term effects of stimulant drugs suggests a more guarded appraisal of their safety.

In a double blind, placebo-controlled crossover study,[15] researchers compared the reactions to Ritalin and placebo of 206 children between the ages of five and fifteen. Treatments were rotated on a weekly basis, and side effects were evaluated using the Barkley Side Effect Rating Scale. Overall response to Ritalin was quite poor, with just 62 percent of children demonstrating an improvement in their behaviors. Five side effects increased significantly with Ritalin therapy: appetite disturbance (19 times more likely than placebo); dizziness (8 times more likely than placebo); stomachache (7 times more likely than placebo); headache (5 times more likely than placebo); and insomnia (three times more likely than placebo).

A study of ambulatory patients seen at the Melbourne Royal Children's Hospital[16] between 1995 and 1996 compared side effects experienced by ADHD diagnosed children (ages 5 to15) in a double-blind crossover trial. Subjects were randomized to Dexedrine (d-amphetamine) or Ritalin (methylphenidate) treatments for two weeks at a time; then, after a 24-hour drug washout period, they were

continued for another two weeks on the other drug. Favorable response rate, as assessed by parents, was 69% and 72% for Dexedrine and Ritalin, respectively. Appetite reduction was a prominent side effect of both drugs. Insomnia was worsened by Dexedrine, but not by Ritalin. The investigators concluded that their results were fairly consistent with the side effects studies conducted by other researchers:

Barkley (1990) 83 children on Ritalin for 7–10 day study, side effects included insomnia, decreased appetite, headache, stomachache on both low and high doses of the drug

Borcherding (1991) 48 children on Ritalin and Dexedrine in crossover study, 83% experienced significant (mild to moderately severe) side effects, including decreased appetite, sleep disturbance, unhappiness

Millichap (1967) reviewed 15 different Randomized Control Trials of stimulants, 15–70 % of Ritalin patients experienced adverse effects, 5–100 % of Dexedrine patients experienced adverse effects

For many patients, it appears to be true that the somatic effects of stimulant therapy—such as appetite disturbance, headaches, and dizziness—may be mild and short-lived. However, the same cannot be said about endocrine, cognitive, and behavioral effects.

Endocrine effects of stimulants

The negative effects of stimulant drugs upon growth rates have been recognized for years. However, some researchers have inappropriately blamed the patient—suggesting that ADHD itself is the cause of growth retardation in stimulant-treated children. The evidence against this argument is abundant. In a recent study conducted by physicians in Australia,[17] none of the 52 ADHD diagnosed children treated in their practice were 'growth deficient' *until* stimulant therapy was initiated. Animal studies have confirmed the growth-impairing effects of Ritalin (methylphenidate).[18]

If anything, the most recent research has confirmed a far too cavalier attitude among physicians with regards to the growth suppressing effects of stimulant drugs. In the Australian study,[17] researchers found that stimulants were associated with progressive declines in both height and weight in 86 percent of their subjects. The decreases were especially prominent in the first 6 to 18 months of treatment but they did not stop throughout the course of treatment. (One patient, who stopped taking dexamphetamine for six months to see if it was still needed for behavioral reasons, experienced a fairly rapid weight gain of 16 pounds but no catch-up in height for another twelve months.)

Similarly, researchers at Yale University in a study involving 84 ADHD diagnosed children (ages 5–17) followed in two large pediatric practices, compared

the growth rates of Ritalin subjects against their own unmedicated siblings.[19] Subjects had to have taken Ritalin for at least two years without interruption. Siblings had to be healthy, born within three years of the patients, and living within the same household. Using height standard deviation scores to compare subjects, the researchers detected significant effects of Ritalin upon mean height and growth velocity: 76 percent of the males and 90 percent of the females experienced significant height suppression after three years of therapy. These effects did not reverse or stabilize at any point during treatment. Growth suppression occurred over a broad range of doses, with boys and girls experiencing an overall height deficit of approximately 3–4 cm (1.2–1.6 inches) over three years.

What is most concerning about these findings is not only the growth suppression, but the fact that neuroscientists and physicians do not yet understand the mechanism which causes it. Many theories have been advanced, but all of them have weaknesses due to inconsistent evidence from clinical and physiological investigations.[20]

At least one research group[21] has detected a possible explanation for stimulant-induced height suppression in the *target* of growth hormone, rather than in the complex endocrine systems (hypothalamus/pituitary/adrenal axis) which control its synthesis and release. Investigators examining the effects of several stimulants (pemoline, methylphenidate, and methamphetamine) upon cartilage discovered that all three drugs inhibited the uptake of sulfate and impaired the formation of glycosaminoglycans. Although these findings have not yet been verified *in vivo* (live humans), they offer an important mechanism through which stimulants may impede the linear growth rates of children. Nevertheless, the possibility still remains that stimulants are disruptive to the regulation of *growth hormone homeostasis*. This has not yet been consistently disproven, and quite a few studies[22] *have* detected associations between stimulants and deficiencies in growth hormone and other growth-related proteins (such as IGF-1 and growth hormone binding protein). To the extent that they disrupt the body's formation of trophic factors or the cellular responses to them, it remains possible that stimulant medications exert an equally disruptive effect upon the *development of the human brain,* as they do upon the developing skeleton.

Cognitive effects

Most discussions of stimulant drugs emphasize their short-term effects upon cognitive functioning. Among the favorable effects noted by researchers and patients are improvements in sustained attention, reaction times, time on task, ability to switch mental sets, academic productivity, impulse control, and certain aspects of learning. Most long term studies, however, have documented a *waning of drug benefits over time*, either because of tolerance to the effects of the drug; changes in the underlying condition (e.g., natural maturation of the brain, decrease in situational demands, or modification of stressors), or direct toxicities exerted by stimulants upon specific aspects of brain physiology and human performance.

The results of neuroimaging studies may provide an explanation for these long-term changes. In the 1980s, a team of researchers[23] performed computed tomography (CT) scans on twenty-two young males (mean age: 23.2 years) who had been diagnosed with ADHD and treated with stimulants in childhood (mean age at diagnosis: 8.7 years). The results of their head scans were compared to the findings of twenty-seven slightly older males (mean age: 28.7 years) with no history of neuropsychiatric difficulties. Even when compared to these *older* healthy controls, the stimulant-exposed subjects demonstrated significant sulcal* widening (58 percent of the ADHD group vs. 3.8 percent of the controls) and cerebellar atrophy** (25 percent of the ADHD group vs. 3.8 percent of the controls). The investigators concluded that 'more research was needed' to determine if the cortical atrophy detected in these ADHD subjects was the result of stimulant therapy or an underlying neuropathology.

In 1994[24] a team of investigators published results from a neuroimaging study involving five healthy males between the ages of 21 and 40. Participants were injected with intravenous doses (0.5mg/kg) of methylphenidate (Ritalin) followed by PET (positron emission tomography) scans 5–10 minutes and 30 minutes after injection. The goal of the study was to identify the effects of Ritalin upon cerebral blood flow. Findings were significant for consistent, *global reductions in blood flow* compared to baseline: 14–36 percent reduction at 5–10 minutes, 10–30 percent reduction at 30 minutes. Because these changes were seen throughout the brain (unlike the regional effects detected by neuroimaging techniques which focus upon glucose metabolism), the researchers surmised that the findings reflected direct effects upon the cerebral vasculature rather than neurons. The investigators concluded:

> Though CBF [cerebral blood flow] changes after oral MP [methylphenidate] are probably smaller than with intravenous MP, its pharmacokinetics may be slower, and CBF decrements may last longer. The extent to which prolonged decrements in CBF with chronic MP occur needs to be evaluated.[25]

As of 2004, no such follow-up investigation had been performed. However, the lead author of this earlier study has reported that he has received funding from the National Institutes of Health for a project to address the 'long term' (one year) effects of oral methylphenidate upon the dopamine system of drug naïve ADHD subjects.[26]

Sophisticated experiments have been developed to evaluate the functioning of different hemispheres of the brain. In one such experiment[27], the responses of

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*sulcal: pertaining to sulci, or invaginations of the brain surface; these furrows appear to widen as underlying brain tissue shrinks or recedes

**cerebellar atrophy: shrinking of the cerebellum, which is a posterior brain structure involved in balance, coordination, and cognitive functioning

26 ADHD children (ages 8–15) to tachiscopic tasks were compared under Ritalin and placebo conditions. (Tachiscopy is a test of visual field dominance which evaluates the speed of processing based upon the appearance of stimuli—dots or digits—to the left or right of a central fixation point.) Reaction times to stimuli producing left visual field advantages (right hemisphere processing) were slower than to stimuli producing advantages in the right visual field. The possibility that stimulant medications might provoke deficits in right hemisphere functioning has been noted by many other researchers, as well.

The concern is that the ADHD literature has been so focused upon the left hemisphere effects of stimulants (such as verbal tasks and sustained attention) that the other half of the brain has been forgotten. *Right hemisphere deficits* have been linked to reductions in diffuse attentional mechanisms, emotional intensity, social responsiveness, and mood (depression and anxiety). Following right hemisphere lesions (such as strokes), patients may develop a 'semantic pragmatic disorder'[28] characterized by an impaired capacity to express themselves in language; flattened intonation; and an inability to perceive emotion, metaphor, humor, and subtleties in the world around them.

If stimulants induce a similar lateralized disorder in children, it is understandable that many ADHD subjects could experience the onset or worsening of deficits in eye contact, playfulness, spatial awareness, the perception of social cues, the use of imagery, the control of impulsive responding, and the holistic integration of feelings, context, and interpersonal relationships—all of which are predominantly right hemisphere functions.

The effects of stimulants upon driving and flying performance have raised additional concerns about their safety. While stimulants have long been used by pilots in the US military for their alerting properties during long term missions, the self-administration of 'combat amphetamines' has provoked criticism in the aftermath of serious accidents involving medicated pilots. In the summer of 2002, two Air National Guard pilots mistakenly bombed and killed Canadian troops over Afghanistan, claiming that 'go pills' consumed before the mission had impaired their judgment.[29] According to a retired Navy Admiral,[30] 'the better warrior through chemistry field' is the focus of aggressive research. While Pentagon officials believe that the 'capability to operate effectively, without sleep' will 'fundamentally change current military concepts of operational tempo[31],' critics believe that it is hazardous to manipulate human sleep schedules artificially. Of particular concern in jet and bomber pilots is the possible induction of stimulant psychosis (hallucinations or delusions), as well as neurological side effects such as tremor, blurred vision, and dizziness.[32]

The topic is pertinent for children and adults with ADHD, almost all of whom receive treatment with stimulants. Several investigations conducted in the US and Canada have documented higher rates of motor vehicle accidents among ADHD drivers, relative to non-ADHD controls. In a study which evaluated the driving records and knowledge of 25 young adults with ADHD,[33] investigators found that ADHD subjects had no deficit in their understanding of road rules.

However, ADHD drivers were more likely to have had their licenses suspended or revoked; more likely to have received repeated traffic citations (mostly for speeding); and more likely to have experienced car crashes while driving (four times more accidents than controls). Although the investigators attributed the accidents and reckless driving styles to the underlying condition of ADHD, rather than to its treatment, the experience of the US military should be instructive for more critical observers. Whatever else one concludes about ADHD drivers, it seems obvious that stimulant medications often fail to control impulsive habits and possibly exacerbate errors in judgment in the same way that amphetamines have been found to impair the performance of combat pilots.

Stimulant drugs and addiction

Beginning in the 1960s, central nervous system stimulants came under increasing regulatory control due to concerns about illicit manufacture and distribution. In 1971, the World Health Organization classified methylphenidate (Ritalin) as a Schedule II drug, due to its high abuse potential. Concerns about Ritalin abuse were relatively moderate for two decades. Curiously, production of the stimulant soared from less then three tons in 1990, to more than thirteen tons by 1997.[34] These developments were alarming to the international community. In 1996 and 1997, the World Health Organization issued press releases (and several letters to the US Drug Enforcement Agency) about the exponential rise in Ritalin, noting that the United States was responsible for 90 percent of the drug's production and consumption. The International Narcotics Control Board (INCB: the World Health Organization agency responsible for monitoring the production and use of controlled substances) explicitly identified a number of concerns about these American developments, including the dangers of inappropriate diagnosis of ADHD; widely divergent prescribing patterns; off-label prescribing to children under six; and excessive duration of treatment (many countries restrict Ritalin use to three years). The INCB was especially worried about the expanding black market for Ritalin, based upon increasing evidence of abuse by individuals who confiscate pills prescribed to others.

Despite the real-world concerns of regulatory authorities, the psychiatric literature has consistently minimized or ignored the addictive potential of psycho-stimulants. More alarmingly, the recent publications of several research teams have suggested that ADHD children should be *encouraged* to use stimulants, in order to *prevent* the emergence of cocaine or other substance dependencies.[35 36]

There are two critical points to be made about psycho-stimulants and addiction. First, although it is uncommon, it is possible for ADHD children to become directly dependent upon their prescribed therapies. Fortunately, this addictive liability appears to be rare as long as the drugs are consumed orally (rather than intravenously or intra-nasally) and in the amounts prescribed. Second, it is possible that stimulants change the human brain over time, so that the vulnerability to a variety of addictions is enhanced. A number of animal studies have documented the capacity of stimulant medications to *sensitize* the brain to

cocaine.[37–39] The precise neuro-physiological mechanisms have not been identified, but hypotheses include the down-regulation of the dopamine transporter (a posited cause of craving), changes in post-synaptic dopamine receptor function or density, or the induction of genes and proteins (such as delta c-fos) which modulate long-term changes in neuronal activity.

If it is true that stimulant medications *prevent*, rather than *induce*, substance abuse, it should be possible to detect that link in epidemiological studies. A large-scale, prospective study of just this kind was performed with 492 ADHD children in Northern California[40] beginning in 1974. After two decades of follow-up, researchers discovered that stimulant-treated subjects developed higher rates of cocaine and nicotine dependence than unmedicated peers diagnosed with either ADHD or behavioral (conduct) disorders:

Percent of Subjects Developing Addiction

	ADHD No stimulant exposure	ADHD Up to 1 Year of stimulant drug	ADHD 1 year or more of stimulant drug	Behavior Disorder no stimulant
	n = 81	n = 9	n = 84	n = 41
Tobacco	32.1%	38.5%	48.8%	32%
Alcohol	32.1%	33.3%	45.2%	39%
Marijuana	22.5%	23.1%	32.1%	34%
Cocaine	15.0%	17.9%	27.4%	12%

A second prospective study,[41] conducted by a different research team, followed 147 hyperactive children (diagnosed between ages 5–12) for approximately thirteen years. The subjects were interviewed at age fifteen (78 percent follow-up) and again in early adulthood (mean age 21, 93 percent follow-up) to explore the use of various substances in relation to ADHD and its treatment. *Childhood exposure to stimulant therapy* was significantly associated with higher experimentation with cocaine (26 percent of stimulant treated vs. 5 percent of unmedicated subjects) by early adulthood. *Adolescent exposure to stimulant therapy* was significantly associated with greater frequency of cocaine use as young adults. Even after controlling for the severity of ADHD symptoms and the lifetime prevalence of conduct disorder features, the researchers detected a statistically significant relationship between prescribed stimulants during high school and a higher rate of cocaine experimentation ('ever use') by adulthood. Despite the limitations of this study (possibly underpowered, no consideration of association between stimulant medication and conduct disorder, and questionable validity of the assessment instruments and data analysis), several findings were consistent with the hypothesis of stimulant sensitization as a risk factor for illicit drug use.

Must we create cybernetic children?

Although Paul Virilio has been described by critics as overly pessimistic about the consequences of technology, his writings provoke a meaningful reflection about the speed of life in relation to man's creations. Virilio proposes that there is a fundamental need within our species for movement through space and time. He worries about the consequences of the Transportation and Information Revolutions, attributing to them the progressive reduction of the human organism to a Brownian-motion existence within a cyberspace pseudo-reality.

Only recently has research within the fields of neuroscience and cognitive psychology caught up with the musings of social theorists like Virilio, expanding upon his ideas about machines and their effects upon the human body. Research on the impact of television, video games, and the Internet suggests that electromagnetic and digital technologies are re-wiring the brain, particularly during the most critical periods of neurodevelopment: childhood and adolescence.

As children have become increasingly exposed to a world of multi-tasking, immediate gratification, and electronic inter-relatedness, their behaviors have come to reflect varying degrees of fitness to the real-time, three-dimensional environments around them. Regrettably, the response of many societies has been a call for the medical control of children, illustrated by the exponential rise in the use of stimulants. By ignoring or minimizing the dangers of these drugs, clinicians have contributed to the suppression of children's growth; the blunting of certain cognitive capacities; and the induction of brain changes associated with higher risks of substance abuse. As childhood continues to be transformed by electrochemical shaping and pharmacological degrading, Virilio's observations become ever more salient. Lived time is changing, to be sure, but there is still time for society to re-evaluate the forces and decisions which have led to the creation of cybernetic children.

Endnotes

This chapter summarizes a presentation at the Tavistock Clinic in London on June 5, 2004 at a conference entitled: *The Construction of Childhood and the Destruction of Children*. The author is grateful to the Psychotherapy Section of the British Psychological Society for the invitation to speak.

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