The Unavoidable Biases of ADHD

This argument takes as its reference the definition of ADHD provided in the DSM-5; we refer the reader to this classification because of its authoritative status in clinical and pharmacological research, i.e. in randomized controlled trials (RCT) used in epidemiology, but also increasingly – and sadly – in clinical practice.

Why do we raise this question?

There are a number of combined reasons why we think that the question of the biases inherent in ADHD is worth discussing.

First of all, for the last twenty years, the number of children diagnosed with ADHD has been rising steadily in all Western societies and especially in the USA. The latest available figures show that 11% of American school-aged children are thought to be suffering from ADHD (1) and there has been a similarly sharp rise in the United Kingdom as well. In France there is no recent comprehensive data available, but all seems to suggest that here, too, prevalence has risen significantly (2). After a delay caused by the reticence of many child psychiatrists with psychopathological training and by the legal restrictions on first-line prescription, the false epidemic is now in full swing. Concurrently with the rise in the diagnosis of ADHD, we have also seen a gradual increase in the consumption of drugs such as methylphenidate. For children, ADHD now represents one of the most common reasons for psychiatric consultation.

In addition, the ADHD diagnosis concerns a growing number of adolescents and adults, especially in its form of an attention deficit with an increased risk of misusing prescription drugs as “boosters” or recreational drugs: these are used at various occasions such as exams and interviews, to deal with professional issues or enhance other types of performance, or as a form of teenage rebellion.

Also, due to the growing systematization of the medical response to a large number of highly varied clinical situations, we see a chronicization of ADHD. Medication is not a cure – it only helps manage symptoms, and since there are no reliable criteria to determine when it should be stopped, we sometimes see situations in which the teenagers or adults wishing to stop because they feel ready to do so become the target of moralizing and guilt-provoking pressure from those around them – “it’s not the right time, it’s too risky, it’s not in your interest, etc.” — or even from clinicians who wish to prevent the “presumed predictive link between ADHD in children and antisocial or addictive behavior in adults” and who rely on

questionable studies that mistake forecasting for prediction (4). This chronicization carries with it a great risk because although the results of RCT looking at the long-term effects of methylphenidate are contradictory, some are indeed worrying (5). After the false positives, we should now be concerned about iatrogenic disability!

Lastly, ADHD appears to be symptomatic of the "secondary effects" of the extensive and exclusive use of DSM's diagnostic method, i.e. overmedication, overdiagnosis and overprescription. These consequences have raised criticism from a growing number of mental health professionals and in particular from the "Initiative for the Clinic of the Subject STOP DSM"(6).

1.1) The bias of non-scientificity. Does ADHD exist? What are its scientific and neurodevelopmental foundations?

In 2002, more than 80 researchers and clinicians from all over the world who had become worried about the worsening media portrayal of ADHD signed a "Consensus Statement," which basically argued that failing to acknowledge this pathological entity was "tantamount to declaring the earth flat, the laws of gravity debatable, and the periodic table in chemistry a fraud." In other words, not recognizing ADHD as a mental pathology meant adopting a reactionary and anti-scientific position. Let us look at their argument more closely.

Can we say that there is currently a scientific basis to support the ADHD diagnosis?

A great number of studies have been looking for the biological causes of ADHD, especially in terms of a disturbance of the dopaminergic system or catecholamine levels. For example, a study conducted on children aged 6-12 years and comparing those labeled with ADHD with healthy individuals showed a higher dopamine transporter density in the basal ganglia and, compared to the control group, increases in dopamine binding in children diagnosed with ADHD. However, the research did not reveal any correlation between increased binding and the gravity of clinical signs and, most importantly, its results were undermined by significant methodological flaws, such as its very limited sample (six children). Similar shortcomings apply to other studies. In fact, the theory of dopaminergic causation or an anomaly affecting another neurotransmitter has never been proved, only correlations exist.

Brain imaging, functional MRI. Several studies have shown anomalies in subjects diagnosed with ADHD, such as a reduction in the overall brain size, reduction of the globus pallidus, changes in the morphology of the corpus callosum or the cerebellum; however, here, too, the results have since been contradicted by other studies, samples were too small and patients had been medicated prior to the administration of methylphenidate. Plus, there have been no

studies including younger and more severely affected children, mostly because of the need to remain still for long periods of time during the exam. Crucially, the results are difficult to interpret because of numerous confounding factors. Lastly, when thinking about functional neuroimaging, we must keep two essential principles in mind:

1. 1) We must distinguish between correlation, identity and causality.

2. 2) A given type of behavior cannot be equated with either a specific brain region or a precise cognitive structure (7).

Genetic research. This type of research relies essentially on studies of twins, comparing the correlation of ADHD in identical twins (MZ) with same-sex fraternal twins (DZ), in order to calculate a rate of heritability. The difference is significant: 75% among MZ twins and 35% among DZ twins. However, these studies contain a structural bias in the sense that they are all based on the so-called "equal-environment assumption," i.e. the hypothesis that co-reared identical twins and co-reared same-sex fraternal twins experience their environment as similar to an equivalent degree. This is clinically absurd because in the case of identical twins the environment (educational, emotional and psychic) is experienced much more similarly than in the case of fraternal twins, especially in terms of the much stronger emotional bond and attachment, as well as a greater level of identity confusion. As to the argument that the difference in the way environment is experienced may be genetically determined, this is simply circular reasoning. We can therefore conclude that there has been no decisive evidence in favor of the genetic origin of ADHD (8).

Can ADHD be connected to an impairment of cognitive processing?

Two main models have been put forth to explain ADHD. Barkley's model revolves around the hypothesis of ADHD representing an impairment of executive functions, a deficit in response inhibition, which has three main components: (a) inhibition of the initial prepotent response to an event (b) interrupting an ongoing response and (c) inhibiting interference likely to arise after the inhibition of the initial response. This inhibition deficit has repercussions for memory, self-regulation of affects and internalization of speech, hence the subject's difficulty in using language to shape moral reasoning and adapt one's behavior to rules; finally there is a deficit of reconstitution, which leads to reduced narrative and creative capabilities.

This theoretical model, which has been supported by the results of a number of trials, is nonetheless unsuited to many complex clinical situations and suffers from two main biases: firstly, cognitive studies do not compare the test subjects with a control group of children

who are younger tha ADHD children but have the same level of cognitive performance, a method well established in neuropsychology; secondly, the executive functions (goal-setting, planning, strategy development, deciding, dealing with information, etc.) lack a precise definition and there is no consensus as to their complete list.

The second model, formulated by Sonuga-Barke, is very similar, in that it, too, considers ADHD a disease affecting inhibition, albeit not as a consequence of an impairment of executive functions, as it is the case in the first model. Other models have been put forth; however, none really corresponds to the questions raised by the often-complex phenomena encountered in clinical work. Moreover, these are only models, not proven etiological explanations.

Based on this brief review of the current state of knowledge, we are led to conclude that so far, the concept of ADHD has relied on assumptions at best, but definitely not on proven scientific facts. The term “neural development” that is constantly used in discussions around ADHD, with the latter supposedly being a neurodevelopmental disorder, has no precise scientific meaning, except as a way to anticipate or speculate on data obtained in the future; it is a hold-all term, a chimera that “sounds scientific and suggests organic causality” and which strives to identify psychopathology with neuropathology.

II. II) The Epistemological Bias of ADHD

Confounding a number of behavioral manifestations (agitation, impulsiveness, lack of attention), as they have been identified by different observers, with a diagnostic entity, instead of seeing them as mere symptoms, has nothing to do with the scientific method; rather we are making a judgment of existence, perhaps with a hidden premise. To claim, based solely of their existence according to a given set of criteria, that these behavioral manifestations constitute an entity whose cause should be the object of research represents in our understanding an epistemological bias, in the sense that we postulate the existence of an entity and then search for its causes through circular reasoning. Behavioral manifestations which are indeed real in their existence (symptoms do exist and people do seek medical help for them) are simply isolated and assumed to constitute a pathological entity with an alleged cause; however, if there is a cause, then there is indeed a manifestation and thus the presumed entity and its alleged cause mutually reinforce each other as if through mythical reasoning. In other words, we were right to isolate the symptoms because there is a cause we are trying to look for, and since there is a cause to be found, these isolated symptoms in fact constitute a disease. In reality, the hidden premise is that there is necessarily an organic cause and that we should only consider manifest symptoms, thus restricting clinical research to physiopathology. However, as of now, no convincing results have been found – perhaps the

reason is not simply time, but in fact precisely this epistemological bias… (9).

II. III) The bias of the method leading to the diagnosis of ADHD

How is ADHD diagnosed? What is the diagnostic method used?

The following is a fictional description of a standard situation frequently encountered in our practice in child psychiatry in France.

Let's begin with a few general remarks:

Today, the process of diagnosing ADHD is in many cases a social and dynamic process, which often involves different stakeholders of the medical, social, educational and legal field, as well as parents' associations.

First of all, we should mention the existence of "pre-diagnostic filters" – a term designating certain professionals (e.g. teachers), media featuring stories about ADHD, and finally parents' associations. These associations are very important in aiding and directing both public health policy decisions and families dealing with an ill child. They undoubtedly play an essential and useful role; however in regard to ADHD, we cannot accept the idea that the "recognition of the validity of ADHD" could be obtained solely on the basis of socio-political organizing, medical activism or by lobbying political authorities, instead of using scientifically verified arguments. Reading the great number of existing testimonies by families and parents, we are moved and struck by the difficulties they had to face and the obstacles they had to overcome. The diagnostic wavering they encountered is unacceptable, but the reasons for it are complicated – a combination of ignorance of certain professionals and the lack of adequate care currently on offer. We must absolutely deal with this situation without trying to deny it, but we must also refuse to fall into the kind of binary Manichean reasoning that divides the world into the "good" clinicians who admit the existence of ADHD and treat children according to scientific criteria, and the "bad" who refuse the diagnosis and treat children and their families either incorrectly or not at all.
We must again emphasize that parents are never responsible for the illness of their child and there is no question of accusing them (of course I am not speaking about cases of attested child abuse). At the same time, being able to tactfully obtain information about, for example, their psychic functioning can be of considerable help in mitigating the difficulties of parent-child interaction and can help us guide parents better than the kind of general advice usually dispensed, which only concerns conscious and manifest behavior (parent training).

Should professionals who refuse ADHD as a diagnostic category offer parents a diagnosis?

The fact is that in many cases this can have a soothing effect and put an end to the feeling of wavering and lack of understanding. What was originally a psychiatric diagnosis has now acquired a wider social meaning, with many important repercussions: it contributes to acceptance and social identity, to the creation of new databases and social benefits, an to socialization throught patient's groups and forums. At the same time, the absence of a diagnosis becomes synonymous with incompetence, the label no longer being purely negative and stigmatizing. There is no general answer to the question of whether a diagnosis should be given or not, but it may be necessary to give one on a case-to-case basis, without diluting this announcement in general information, but instead explaining, using ordinary understandable terms, what this means according to the clinician, what it will mean for the child and the family, as opposed to what is commonly understood as ADHD.

Now let us look at the biases inherent in the "diagnostic technique" itself, one that is employed by clinicians following the recommendations of the DSM-5 and others involved, such as parents, teachers or special-needs tutors.

First of all, the diagnosis of ADHD relies on the identification of certain behavioral signs such as hyperactivity or lack of attention. These are non-specific signs that can be observed in many pathological conditions, for example: mental retardation, epilepsy, intoxication, child abuse, brain lesions, severe anxiety, learning disorders, problematic family situation or simply immaturity, etc. These signs may appear due to various reasons, with either biological or environmental — and often mixed — origins. Furthermore, the child's symptoms often tend to fluctuate over time and as the child develops; this volatility can sometime be interpreted as comorbidity: the child has "oppositional defiance disorder" at one time and "mood disorder" or "anxiety disorder" at another. This creates a lot of confusion in randomized clinical trials because ADHD can be associated with signs related to the spheres of affects, personality or communication. When using the DSM-5 categories, differential diagnosis can turn out to be a delicate question.

These behavioral signs have therefore no pathognomonic value because they are not pathological in themselves – any child can be easily distracted, have a short attention span or become agitated. They vary from one individual to another, from one context to another; there are tolerance levels and situational effects, but a tolerance threshold is not a biological threshold. Therefore we are trying to measure the excesses; however, in terms of excess there can a great variation of appreciation – the inclusion thresholds are not objective. For example, tolerance to hyperactivity is probably not the same in a large countryside home and in a small city apartment. Normal variations should not be mistaken for pathology.

In other words, despite the effort to establish a detailed description and a restrictive

framework, the diagnosis of ADHD in DSM-5 contains much vagueness and subjective judgment, which constitute a bias even according to the principles of DSM-5 itself.

There is another source of bias in the diagnostic procedure, which is connected to the fact that the diagnosis does not generally rely on either listening to the patient or on standardized tests, but instead on more or less structured interviews. Clinicians have several interview models at their disposal. The most commonly used is the Conners test, which includes around fifty items and comes in two different versions: one for parents, the other for teachers. In practice, these tests have drawn a great deal of criticism, such as that the form and definition of the items is not always clear; they often use negative forms which leads to anomalies; there is a lack of descriptive characteristics, resulting in confusion which hampers the quality of medical response; some items refer to different types of behaviors which cannot be equated with one another, there is poor reliability between the results of successive tests, etc. We are indeed very far from the objectivity of biological markers.

In conclusion, the diagnosis of ADHD is biased in a number of ways and public authorities must urgently address the problem. It is paradoxical to hear those who accuse French child psychiatrists of not paying enough attention to what is happening elsewhere in the world and especially in the U.S., while they themselves pay no attention to what is happening in the world and in the U.S. in terms of the ADHD epidemic, with some describing the spike in prevalence as a national catastrophe. What is there to be done? (10)

Here are just a few possibilities:

We must stop treating the DSM-5 as the sole clinical reference – clinicians must be able to choose. Adopting an approach based on the idea of psychic structures or categories is more sensible because the hyperactive syndrome, if indeed it should in all cases be treated, does not have the same consequences and lead to the same changes in both psychic structures (neurosis or psychosis) and does not require the same treatment. Hence there is a need to teach psychopathology alongside neuroscientific disciplines, to train clinicians to be able to distinguish between complex clinical situations, to promote CFTMEA, the French classification of child and adult mental disorders, in parallel to DSM-5 because the former follows the distinction between different psychic structures.

Indications for the prescription of medication must be made more specific and the use of pharmaceutical drugs as sedatives or to stabilize the patient’s situation must not lead to chronicization (11). It is paradoxical that those who habitually accuse psychoanalysts of “blaming the parents” can themselves blame teenagers and young adults wishing to stop medication, using the precautionary principle as their argument. The criteria of when and

how should the use of methylphenidate be stopped must therefore be made as specific as possible.

Public authorities must stop gulping down the discourse on “the brain” and its social uses and instead keep to real scientific advances; they must pay attention to clinical experts who are truly open to science and who work unhindered by conflicts of interests. The infernal machine sustained by powerful interests and fueled by DSM-5 is already in motion – we still have some time to put on the breaks, but it is quickly running out.

References:

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