Autistic-Undisciplined Thinking in the Practice of Medical Trichology

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ABSTRACT

Medical trichology is the branch of dermatology that deals with the scientific study of the hair and scalp in health and disease. As any discipline, the practice of medical trichology is not immune to malpractice, either deliberately or carelessly. In his publication “Autistic Undisciplined Thinking in Medicine and How to Overcome It,” Swiss psychiatrist Eugen Bleuler describes yet another form of malpractice in medicine reflecting autistic-undisciplined thinking. Autism is not limited to psychopathology, but inherent to the thinking of man throughout history in his drive for knowledge, with thousands of theories lacking any basis in reality. Bleuler recognized the drive character of autistic thinking and how it leads to conclusions that are unshakable because they are determined by, and fulfill, emotional needs, rather than rational argumentation. Even with correct questions, the complexity and incalculability of some problems are often so great that it cannot do justice to realistic thinking and the boundaries between inadequately substantiated hypothesis and autistic sham explanation disappear. Statistics, careful research design, and the attempt to impose stringent methods on our thinking are to be commended. The habituation of the public to useless medicine, to misconceptions, is not hygiene, but negligent endangerment. It has created an industry that largely lives on the autistic thinking of patients and doctors and because it is prosperous, makes propaganda among lay people as well as among doctors that necessarily leads to abuses. This article aims at exposing the most prevalent among abuses in trichological practice due to autistic-undisciplined thinking, specifically: iron supplementation, antiandrogenic treatment, and individualized cell-based therapy in female androgenetic alopecia, treatment of folliculitis decalvans with retinoids, and the value of nutritional therapies.

Key words: Antiandrogens, autistic-undisciplined thinking, Eugen Bleuler, folliculitis decalvans, iron, nutritional therapies, platelet-rich plasma, trichology

INTRODUCTION

“The more our knowledge widens, the less prevalent autistic thinking becomes within common sense.”

-Eugen Bleuler

Eugen Bleuler (1856–1939), Bleuler was convinced that complex mental processes could be unconscious, and he inspired his personnel at Burghölzli to study unconscious and
psychotic mental phenomena. He introduced the term schizophrenia in 1908 in a publication based on the observation of 647 Burghölzli patients, and is also credited with the introduction of a concept fundamental to the understanding of schizophrenia: autism, denoting the loss of contact with reality, frequently through indulgence in bizarre fantasy. Bleuler was celebrated for his skillful clinical observation and inclination to let symptoms speak for themselves, as well as for his enlightening writings.

Bleuler’s original monograph, “Dementia praecox or the Group of Schizophrenias” (1911) had a profound impact on psychiatry. His publication referred to here, “Autistic Undisciplined Thinking in Medicine and How to Overcome It” (1919) was a seminal work as well, widely read in Europe at the time, to appear in a complete English Edition only in 1970. In this work, Bleuler points out that autism is by no means limited to schizophrenia, but inherent to the anthropocentric thinking of man throughout history in his drive for knowledge, with thousands of theories lacking any basis in reality.

Autistic thinking has particularly been practiced in medicine, and regrettably is fostered by physicians who delude the patient and themselves. Bleuler comprehended the drive character of autistic thinking and how it leads to conclusions that are unshakable because they are determined by, and fulfill, emotional needs. Bleuler attacks with passion and validity a variety of medical malpractices reflecting undisciplined autistic thinking. While it may be comforting to recognize that our current blunders have historical precedents, it only highlights the magnitude of the problem. If it were easy to forsake this type of thinking and to approach reality more strictly, we would have done so more aptly in the last 100 years. The use of statistics, careful research design, and the attempt to impose more stringent methods on our reasoning are to be commended. Moreover yet, even where we have correct questions, the complexity and incalculability of some problems are often so great that it cannot possibly do justice to realistic thinking, and the boundaries between inadequately substantiated hypothesis and autistic sham explanation disappear.

Different doctors have different styles of practice and different approaches to problems, but all are susceptible to the same types of mistaken in their thinking. Ultimately, errors, and not gaps, prevent science from advancing. A century after Bleuler, Jerome Groopman from Harvard Medical School again focuses on the thinking errors in medicine in his publication “How Doctors Think” (2007). According to his estimation, thinking errors make up for 80% of medical mistakes, whereas only 20% are due to technical mishaps: snap judgment, stereotypical thinking, premature conclusion, and herd instinct are only a few of the subtle traps that dangerously narrow the vision of the physician. Moreover, if we look at the treatment of diseases in any textbooks, we notice how often the recommended remedies are simply enumerated, without any specific indication of why and in what case the one, in which case the other remedy should be chosen, and especially without differential indication. Finally, it is an old but obvious statement that the more remedies for disease are suggested, the more certain it is that none really works; if one had one that heals with some certainty, the others would be disregarded. Instead, trial and error algorithms are proposed, while algorithms discourage physicians from thinking independently and with creativity.

Thinking is inseparable from acting. Inaction is neither what is expected from a physician, nor what a physician expects from himself. Doctors typically prefer to act even when in doubt about the nature of the problem and its management. The tendency toward action rather than nonaction is more likely to happen with a doctor who is overconfident, and whose ego is inflated, but it can also occur when a physician is desperate and gives to the urge to do something, often sparked by pressure from the patient. Ultimately, the trade with fashion treatments, which have no actual justification, shows us yet another negative side. The habituation of the public to useless medicine, and to misconceptions, is not hygiene, but negligent endangerment. It has created an industry that largely lives on the autistic thinking of patients and doctors and because it is very capable and prosperous, makes propaganda among lay people as well as among doctors that necessarily leads to abuses.

**ABUSE IN IRON SUPPLEMENTATION**

While there is no doubt that iron deficiency represents the most common nutritional deficiency, particularly in adolescent girls and women of childbearing age, and iron deficiency is undisputedly a cause of significant morbidity, including hair loss, the various observational studies that evaluated the association between decreased ferritin levels and hair loss have resulted in opposing results. The controversy starts with a debate over what is the normal serum ferritin level for women and is further complicated by the use of different reference ranges by different laboratories, based on individual interpretations of the literature on this subject.
Search satisficing is the tendency to stop searching for a diagnosis once you find something. Finding something may be satisfactory, but not finding everything is suboptimal. It is a natural cognitive tendency to stop searching, and therefore stop thinking when one makes a seemingly significant finding. We value too highly information that fulfils our desires and fail by confirming what we expect to find by selectively accepting or ignoring information. A typical example of this error is reducing the treatment of a female complaining of hair loss to iron supplementation in detected iron deficiency, while at the same time she may be suffering of female androgenetic alopecia, and is on an oral contraceptive with pro-androgenic action, that are the actual culprits. The true expert, though, having learned about bias and search satisfaction, consciously tries to keep his mind open so that he sees beyond his pre-conceptions.

Bregy and Trüeb evaluated the relationship between serum ferritin levels and hair loss activity determined by trichograms in a retrospective case study of 181 women with hair loss who underwent biochemical investigations and trichograms, and found no correlation between ferritin levels and telogen rates, both in women with telogen effluvium and women with androgenetic alopecia, refuting at the same time the common opinion that androgenetic alopecia should have a threshold effect on sensitivity to low ferritin levels.

Therefore, in women with hair loss, the role of tissue iron status within limits regarded as normal has been overestimated.

In fact, there is the possibility that increased iron storage could enhance oxidative injury by inducing the Fenton reaction, with the prospective of increasing the risk of cardiovascular disease and cancer. In an attempt to demonstrate the pro-oxidative capacity of oral iron supplementation, King et al performed a study on women with low iron stores (plasma ferritin < or = 20 μg/L) receiving a daily iron supplement for 8 weeks at a level commonly used to treat poor iron status. They measured increased lipid peroxidation by ethane exhalation rates and plasma malondialdehyde. The women served as their own control as pre- and post-supplementation periods were compared. After 6 weeks of iron supplementation, serum ferritin almost doubled and body iron more than doubled, hemoglobin levels increased slightly, and other indicators of iron status became normal. However, plasma malondialdehyde and breath ethane exhalation rates increased by >40% between baseline and 6 weeks of supplementation. These increases correlated significantly with plasma iron and ferritin levels. The authors concluded that the increased indicators of lipid peroxidation with duration of supplementation and as iron status improved suggest that providing daily nearly 100 mg iron may not be a totally innocuous regimen for correcting iron depletion.

Consequently, the practice of uncritical iron supplementation in women complaining of hair loss is to be rejected both as unnecessary and as potentially detrimental.

**ABUSE IN ANTIANDROGENIC TREATMENT**

Androgenetic alopecia represents the single most frequent cause of hair loss in both men and in women. The condition is understood to be heritable, androgen-dependent, and to occur in a defined pattern, though with significant differences between men and women with respect to age of onset, frequency, and pattern of alopecia.

Of the various hormones that affect hair growth, the most studied have been the androgens, particularly as they pertain to male androgenetic alopecia. Since Aristotle first noted that maleness and sexual maturity were required for balding, it was not until 1942 that Hamilton's observations on men deprived of testicular androgens by castration established beyond doubt that androgens, in the form of testosterone or its metabolites, are prerequisites for the development of male androgenetic alopecia. Ultimately, the report of an unusual form of incomplete male pseudohermaphroditism due to a genetic deficiency of type 2 5α-reductase by Imperato-McGinley et al. in 1974 implicated dihydrotestosterone as the principal mediator of androgen-dependent hair loss, since affected males who were homozygous for the gene mutation did not develop androgenetic alopecia, at the same time establishing effective targeted treatments for male androgenetic alopecia with the respective 5α-reductase inhibitors finasteride and dutasteride.

Since Hammerstein's proposition of the use of antiandrogens to treat women with symptoms of hyperandrogenism, such as hirsutism, seborrhea, and alopecia, antiandrogen therapy was established as a treatment of female androgenetic alopecia as well. Generally, a high dosage reverse sequential therapy of 100 mg cyproterone acetate (CPA) on the 5th–14th days of the menstrual cycle and 40 mcg ethinyl estradiol (E2) on the 5th–25th days was used in severe cases, whereas low dosages of 2 mg CPA and 50 mcg of E2 preparations were used for light cases. Spironolactone is yet another agent with antiandrogenic action considered in the treatment of female androgenetic alopecia where CPA has not
been available. Spironolactone is a competitive inhibitor of aldosterone receptors, which also blocks androgen receptors and increases metabolic clearance of testosterone. Finally, flutamide is a nonsteroidal antiandrogen which is used primarily to treat prostate cancer. It is also used in the treatment of androgen-dependent conditions in women such as acne, excessive hair growth, and alopecia in women.

Meanwhile, observations of androgenetic alopecia before puberty, in patients with hypergonadotropic hypogonadism, and the androgen insensitivity syndrome, though exclusively presenting with a female pattern, have indicated that the role of androgens may have been overestimated in the pathogenesis of female androgenetic alopecia. In fact, female androgenetic alopecia represents a yet poorly understood condition with a more complex etiology and differences from male androgenetic alopecia, with hereditary, hormonal, inflammatory, vascular, and dietary factors, as well as a variety of significant comorbidities: psychological, smoking, obesity, endocrine, and metabolic.

Ultimately, Vexiau et al. compared topical minoxidil 2% and CPA in the treatment of female androgenetic alopecia by randomly assigning 66 women for 12 cycles into two groups, 33 received two local applications (2 mL/day) of topical minoxidil 2% plus combined oral contraceptive and 33 received CPA 52 mg/day plus E2 35 μg for 20 of every 28 days. The investigators found that minoxidil treatment was more effective in the absence of other signs of hyperandrogenism, hyperseborrhea, and menstrual cycle modifications, when the Body Mass Index (BMI) was low, and when nothing argued in favor of biochemical hyperandrogenism, while CPA treatment was more effective when other signs were present and when the BMI was elevated, factors that favored a diagnosis of biochemical hyperandrogenism.

While oral finasteride has unanimously been shown to be effective in the treatment of male androgenetic alopecia, its efficacy in women is inferior. In a double-blind, placebo-controlled, multicenter trial, Price et al. demonstrated that oral finasteride, 1 mg/d, did not slow progression of hair loss or promote hair growth, nor improve follicular counts in horizontal sections of scalp biopsies in postmenopausal women with androgenetic alopecia. One explanation might be that the different pattern of hair loss in the majority of women from that usually seen in men may be due to differences in the relative levels of 5α-reductase, aromatase, and androgen receptors in scalp hair follicles. Trüeb et al. originally reported successful treatment of androgenetic alopecia with higher doses of oral finasteride in 5 normoandrogenic, postmenopausal women, and in the so far largest series of 37 pre-menopausal women treated with finasteride in doses of 2.5–5 mg daily, Iorizzo et al. showed some improvement in 62% as assessed by global photography. Differences in response of women to oral finasteride have led to the suggestion that not all types of female hair loss have the same pathophysiology, i.e., a distinction should be made between alopecia with early (premenopausal) or late (postmenopausal) onset, and with or without hyperandrogenemia. Nevertheless, up to date, no predictive factor for response to finasteride treatment has been identified in women with female androgenetic alopecia.

There are aspects to human biology and physiology that just are not predictable. Doctors, like everyone else, display certain psychological characteristics in the face of uncertainty. There is the overconfident mindset: people convince themselves they are right because they usually are. But biology is inherently variable. One would think that primary care physicians, such as general practitioners grapple most with uncertainty. The truth is that specialization in medicine often confers a false sense of certainty. When physicians shift from a theoretical discussion of medicine to its practical application, they do not acknowledge the uncertainty inherent in what they do. The denial of uncertainty, the inclination to substitute certainty for uncertainty, is one of the most remarkable human psychological traits. It is both adaptive and maladaptive, and therefore guides and risks to misguide. Physician’s denial of awareness of uncertainty serves similar purposes: it makes matters seem clearer, more understandable, and more certain than they really are, ultimately it aims at making action possible. A defense against uncertainty is the culture of conformity. Doctors are not taught to keep an open mind. Rather, they are educated for dogmatic certainty, for adopting one school of thought or the other. A typical example is the practice, or rather malpractice, of antiandrogen treatment for androgenetic alopecia in women without evidence of hyperandrogenemia.

Ultimately, antiandrogen treatment is not without problems. Dose-related side effects of CPA, including weight gain, fatigue, loss of libido, mastodynia, nausea, headaches, and depression, are common. Spironolactone may cause breast soreness and menstrual irregularities, and flutamide may cause fatality due to toxic hepatitis raising the question of ethical issues related to its use for female androgenetic alopecia. Only finasteride is well-tolerated and therefore represents the safer option in postmenopausal and infertile women, whereas in women of childbearing age the risk of teratogenicity forbids its use.
Platelet-rich plasma (PRP) is blood plasma that has been enriched with platelets. It is also marketed as individual cell-based therapy. As a concentrated source of autologous platelets, PRP contains and releases through degranulation several growth factors and cytokines. PRP has gained popularity among physicians attending hair loss patients with a primary commercial interest, though the use and clinical validation of the method for diverse dermatologic conditions are still in the early stages. Results of basic science and preclinical trials have yet to be confirmed in large-scale controlled clinical trials.

A Cochrane review on PRP has been performed in chronic wounds. Even if the conclusion of the respective review was positive, this would have not been surprising, since blood platelets have a physiologic role in wound healing. In contrast, it is more difficult to comprehend the role of platelet-derived growth factors and cytokines for hair growth and treatment of hair growth disorders. Out of this reason, a stringent evaluation of existing data in favor of PRP for treatment of hair-related disorders is indispensable. A more recent systematic review of PRP for the treatment of androgenetic alopecia concluded that existing literature suggests that PRP is a low-risk intervention associated with good patient satisfaction and some objective improvement in outcomes, while further research is needed to optimize preparation and delivery methods as well as standardize measurements of clinical outcomes.

As monotherapy, the efficacy of PRP for treatment of androgenetic alopecia is clearly inferior to the current first-line therapies with minoxidil and the 5α-reductase inhibitors, but PRP may enhance treatment results in combination. From an ethical point of view, the costs in relation to the additional benefit for patients have to be weighed against the financial profit of the doctor.

With regard to alopecia areata, there exists a single, double-blind, placebo- and active-controlled, half-head study to evaluate the effects of PRP. However, in an era when a more comprehensive understanding of the immunologic basis of alopecia areata through genome-wide association studies has opened the venue to more targeted treatments of alopecia areata, specifically the Janus kinase inhibitors, the proposal of PRP with its poorly defined mode of action represents an intellectual step backward.

The scarring alopecias represent a diverse group of disorders that cause permanent destruction of the pilosebaceous unit and irreversible hair loss. They feature some diagnostic and therapeutic challenges: Many have neither known cause nor consistent clinicopathologic findings. There exists an inconsistent use of terminology with different terms to denote same entities and single terms to denote different conditions. Frequently, there is a significant patient’s or doctor’s delay, when irreversible scarring has occurred. The goal of therapy is mostly to halt further progression. Since the causes are unknown in many cases, therapy has remained empiric and nonspecific, while published data on efficacy have low levels of medical evidence. Since structural changes in the course of scarring alopecia are irreversible, there is a clear need for early intervention. With expanding technologies for dissecting the basis of disease, there is hope for a deeper understanding of the underlying pathogenesis for appropriate therapeutic interventions versus the currently proposed trial and error algorithms.

Within the maze of varied conditions leading to scarring alopecia, the most important is to keep a neat nosologic classification in mind, based both on morphology and a pathogenic understanding. Accurate diagnosis based on careful patient history, clinical examination, microbiological studies, and scalp biopsy is prerequisite to treatment.

A typical example of autistic-undisciplined practice in the management of scarring alopecia, despite a deeper pathogenic understanding of the respective condition, is the treatment of folliculitis decalvans with oral retinoids.

Like dissecting cellulitis of the scalp and acne miliaris necrotica, folliculitis decalvans represents a chronic recurrent pustulofollicular scalp inflammation resulting in scarring with a neutrophilic inflammation on histopathologic examination. On transmittal of a scalp biopsy for histopathological examination, even the histopathologist may confound the three entities adding to the confusion. Prerequisite for a correct interpretation of a histopathological sample is that the clinician chooses his words carefully in communication to the laboratory. Appropriate wording can make perception and analysis better. If the physician does not give us a full history, just the one question in his mind, then he will technically tailor the exam to that one question, and risk missing something else that is important. Many dermatologists want a diagnosis and do not read a histopathologist’s description of observations on scalp biopsies.
From a pathogenetic point of view, dissecting cellulitis of the scalp represents a follicular occlusion disorder, just as acne conglobata and hidradenitis suppurativa, while acne miliaris necrotica is understood to represent an abnormal inflammatory reaction to the hair follicle's commensal microorganisms associated with stress-induced scalp seborrhea, which is why both respond to treatment with oral retinoids. In contrast, Matard et al.[23] have provided the evidence for the presence of bacterial biofilms, usually Staphylococcus aureus, in scalp hair follicles to be pathogenic in folliculitis decalvans. The presence of a bacterial biofilm at the interface of the hair shaft explains the high relapse rate of folliculitis decalvans despite even prolonged combined oral antibiotic regimens, while there is no rational for the use of oral retinoids. In fact, Trüeb et al.[24] originally proposed that punch excision of hair tufts or excision surgery of affected scalp areas may provide more sustained results since this would reduce the number of the infected hair follicle with bacterial biofilms. The same applies to laser-assisted destruction of affected hair follicles.[25]

VALUE OF NUTRITIONAL THERAPIES

In his publication “How Doctors Think,” Jerome Groopman stated that “aside from relatively common dietary deficiencies—lack of vitamin B12 causing pernicious anemia, or insufficient vitamin C giving rise to scurvy—little is known about the effects of nutrition on many bodily functions.”[26] The fact is that quantity and quality of hair are closely related to the nutritional state of an individual. Normal supply, uptake, and transport of proteins, calories, trace elements, and vitamins are of fundamental importance in tissues with a high biosynthetic activity such as the hair follicle. Moreover yet, there is hardly another field with so much prejudice, misconception, and debate as diet and health, let alone hair health. It would appear that unless hair loss is due to a specific nutritional deficiency, there is only so much that nutritional therapies can do to enhance hair growth and quality. However, there are internal and external factors, such as aging and environmental oxidative stress, that influence hair health to such a degree, that nutritional therapy can boost hair that’s suffering from these problems, despite the reservations of many physicians who are not knowledgeable in the field of nutrition and health.

In the 1960s, the original studies were performed on a putative role of L-cystine and L-methionine in the production of wool in sheep. It was found that enrichment of even what appeared to be a normal diet with sulfur-containing amino acids increased wool production. When considering which dietary supplements could be used for improving hair growth in humans, L-cystine was, therefore, a candidate. Starting in the early 1990s, studies on the effect of dietary supplements in humans containing L-cystine in combination with medicinal yeast, pantothenic acid, and thiamine were performed for the evidence-based marketing of respective nutrient-based therapies in hair loss.[26] Ultimately, experiments performed on C57BL/6 mice who developed hair loss when exposed to cigarette smoke,[27] demonstrated that this effect could be prevented by the oral administration of N-acetylcysteine, an analog and precursor of cysteine and reduced glutathione, as well as cystine, the oxidized form of cysteine.[28] The effect was interpreted as to be possibly related to the glutathione-related detoxification system. Together with the in vitro observations of Upton et al.[29] that dermal hair papilla cells in androgenetic alopecia were particularly sensitive to environmental oxidative stress, these observations may identify alternative pathways that could lead to complementary nutritional strategies for the treatment of androgenetic alopecia.

CONCLUSION

Despite Bleuler’s optimistic view that “the more our knowledge widens, the less prevalent autistic thinking becomes within common sense,”[30] today’s practice of medical trichology still demonstrates durable traits of autistic-undisciplined thinking.

As a general rule, prerequisite for delivering appropriate patient care is an understanding of the underlying pathologic dynamics of hair loss and the potential multitude of underlying cause relationships. By approaching the hair loss patient in a methodical way, and commencing with objects the simplest and easiest to recognize, and ascending step by step, to the knowledge of the more complex, an individualized treatment plan can be designed. For this purpose, it is wise to divide each of the difficulties under examination into as many parts as possible, and as might be necessary for its adequate solution, and finally to make enumerations so complete, and reviews so general, so that nothing is omitted, that might compromise success.

Ultimately, the errors of careless thinking can be readily given up following these rules, as originally proposed by French philosopher René Descartes (1596–1650) in his groundbreaking “Discourse on the Method of Rightly Conducting One's Reason and of Seeking Truth in the Sciences” (1637),[31] while it remains to fear that even in the 21st century genuinely autistic thinking leads to convictions which are firmly held, in fact delusional, and by definition,
therefore, cannot be overcome, to the detriment of patients and of our medical discipline.[19]

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