Applied Kinesiology and the Factors and Biomechanics of Androgenic Alopecia - A Corrigible Look

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Abstract

Baldness and balding are epidemic problems that affect both sexes at varying ages on almost all levels of being. The subject is a large one. Understanding and correction require the identification of balding types by category, among many things. These include categories related to stress, improper nutrition, hormonal imbalances, and, of late, gravity and COVID-19-a type of stress related hair loss. The types of baldness by common diagnostic name include Androgenic alopecia which includes male pattern balding (MPB), female pattern balding (FPB), and receding hairline. Then other types such as alopecia universalis, alopecia areata, traction alopecia, and central centrifugal cicatricial alopecia (CCCA), In the last few years, we have identified causes of hair loss related to dysfunction of the stress system by way of adrenal, thyroid, and pituitary dysfunction, nutrient deficiencies, chemical toxicity and poisoning of the scalp and hair follicles, emotional trauma, and the focus of this paper, - gradual biomechanical aberrations. It appears that when gradual biomechanical aberrations are identified and addressed, and if stem cells in the hair follicle are properly stimulated and supported, hair regrowth, and the prevention of androgenic alopecia may be possible. Applied Kinesiology tools applied to the cranium and hair follicle allow the identification of causes and the creation of a care plan with a foundation in the restoration of sound cranial biomechanics and function. This means that for the management of improved scalp and hair health to be successful and long lasting, it must include correction and basic maintenance of the biomechanical structures of the cranial vault, osteo-myofascial system, and remaining stomatognathic system.

Key Indexing Terms

Chiropractic, Applied Kinesiology, Male Pattern Balding, Androgenic Alopecia, Hair Follicle Stem Cells, Cranial Fault, Subluxation, Manual Muscle Test, MMT, Myofascial System, Functional Medicine, Galea, Occipitofrontalis, Molding

Introduction

Unanticipated hair loss affects untold numbers of the population of the world and the USA. There are many causes and types of hair loss. The causes include the more obvious gross nutrient deficiencies, traumatic stress, and then, more ambiguous and incomplete, are the various explanations for male pattern and female pattern baldness. This paper will

orient, arrange perspective, as well as validate work already performed on these two subjects and advance them to a new level not seen before, using tools we already own.

Jargon relating to hair loss

Bone remodeling is the process of bones changing size, shape and sometimes growth, holographic subluxation – is a shape based subluxation that does not match the nervous systems expectation of shape; molding – the process of changing shape of the skull intentionally or not; vellus hair – fine hair; lanugo hair – a baby hair; anagen – the first stage of hair shaft development and the growth period; catagen – the disruption of the growth process and end of hair shaft growth and nourishment; telogen- the resting phase of a hair follicle; telogen effluvium – the expulsion of a hair shaft from the follicle, a shedding; Androgenic alopecia – hair loss based on elevated androgens; miniaturization - thinning of hair so that a weak and fine hair shaft grows; dihydrotestosterone –a hormone, biochemically it is reduced testosterone; Superficial Musculoaponeurotic system (SMAS) – fascial system in the head and neck that includes the platysma muscle superior to the clavicle rising to the superficial temporal fascia..

Background and Preparation

A web search on the topic of hair loss provides millions of results. From gels to-lotions-to surgery and implants to wigs, hair is often on many people's minds.
Hair loss affects 80 million Americans, with approximately 60% of men and 40% of women suffering from it. The most prevalent type among the 80 million is androgenic alopecia (AA) (American Academy).

This type includes male and female pattern balding (*Skin Conditions by the Numbers*, n.d.) . Current explanations for male pattern balding follow. In 1987, a seminal study was done on scalp Dihydrotestosterone (DHT) elevation on bald areas of the scalp of men suffering from male pattern balding (MPB). We know that testosterone irreversibly converts to dihydrotestosterone (Caballero).

Testosterone + Alpha 5 Reductase ---------> DHT

Male pattern balding by convention since 1987 was stated to be related to an elevated level of scalp DHT and scalp 5 alpha reductase, the enzyme that converts testosterone to DHT, which causes "susceptible hair follicles" to produce weak hair shafts and then produce none at all. Blood tests for DHT in one study showed no difference between control and studied subjects. To date, there are no blood tests that accurately correlate blood androgen levels with balding (Donovan 2014).

<u>Steroids</u>

The common agreement and background information is that androgens have been determined to target specific hair follicles that have more receptors for 5-alpha reductase and DHT, and more of these are located in the frontal lobe than the occipital (Price 2003). This is the area of male pattern baldness. These androgens are well documented to also be responsible for secondary sex characteristics such as the following in men:

- Pronounced body hair characteristics (beard, chest, etc.) and is usually coarser.

- Heavy musculature
- Angular features (i.e. square jaw, triangular mid region)
- Narrow hips
- Muscular pectorals (chest)
- Overall, there is less fat tissue.
- Deeper voice

In women, these secondary sex characteristics include:

- Less pronounced body hair characteristics (mostly in pubic region, hair all over is usually finer)
- Lighter musculature
- Rounded features (i.e. softer facial features, hourglass mid-region)
- Wider hips (for child bearing)
- More pronounced breasts with more fatty tissue
- More fat tissue overall.
- Higher voice

The following androgens also play a role in hair growth:
Testosterone creates hair growth and secondary sex characteristics that differentiate men from women.

Cortisol is a stress and everyday adrenal hormone that is elevated during stressful times and is catabolic to all tissues. Chronic exposure is also catabolic and gene expression in many cells can change from an acute exposure as an anti-inflammatory to a chronic exposure that stimulates nuclear genomic increases in levels of NfKb, making the inflammation epigenetic or a chronic product of genetic expression instead of acute local irritation.

Cortisol may also work in a feedback loop with DHT. Elevated cortisol levels cause local increases in DHT levels as these are meant to lower the cortisol (Toufexis).

Gravity
Another hypothesis proposed in a 2008 paper is that hair loss is a direct product of the forces of gravity. According to this new theory, the pressure created by the weight of the scalp is the cause of male pattern baldness (USTUMER 2008).

Genetics
Genes are thought to play a role in AA. The problem in each person is thought to be polygenic and a perfect storm between endocrine imbalances and polygenetic factors. Two genes have been identified. We do know that epigentic factors can modify genetic expression. Improper nutrition has been put forth as a major factor in some types of baldness; not solely in the case of AA.

Phases of hair growth

At birth, or in an anorexic adult, we see lanugo hair. This hair eventually falls out after birth and is replaced by fine hair called vellus hair. Vellus hair is present until puberty. After that, a terminal or adult hair replaces it.

Parts of the scalp hair unit

Hair is composed of a shaft – "the hair," which grows from a follicle that has germ cells at the bottom receives its blood supply from vessels in the underlying tissue.

Phases of terminal hair growth

Terminal hair growth occurs in stages of follicle and shaft activity. Four of the most basic stages are listed below (Kiesewetter 1990) ;

- Anagen–lasts 2-7 years and represents the growth of the hair shaft and how long it can become.
- Catagen -the hair shaft shrinks and detaches from its nutrient source – it has a 10 day duration and can be gradual or acute and is impacted by cortisol.
- Telogen - This is the unit's resting phase. The shaft of hair is now detached but still in the follicle. The stem cells have made or are resting but can make a new hair shaft. At any given time, 10-15% of the hair is in this stage. This can last three months or longer.
- Exogen: This is still in the resting phase, but new hair will begin to grow, and the detached hair will leave the follicle and be shed.
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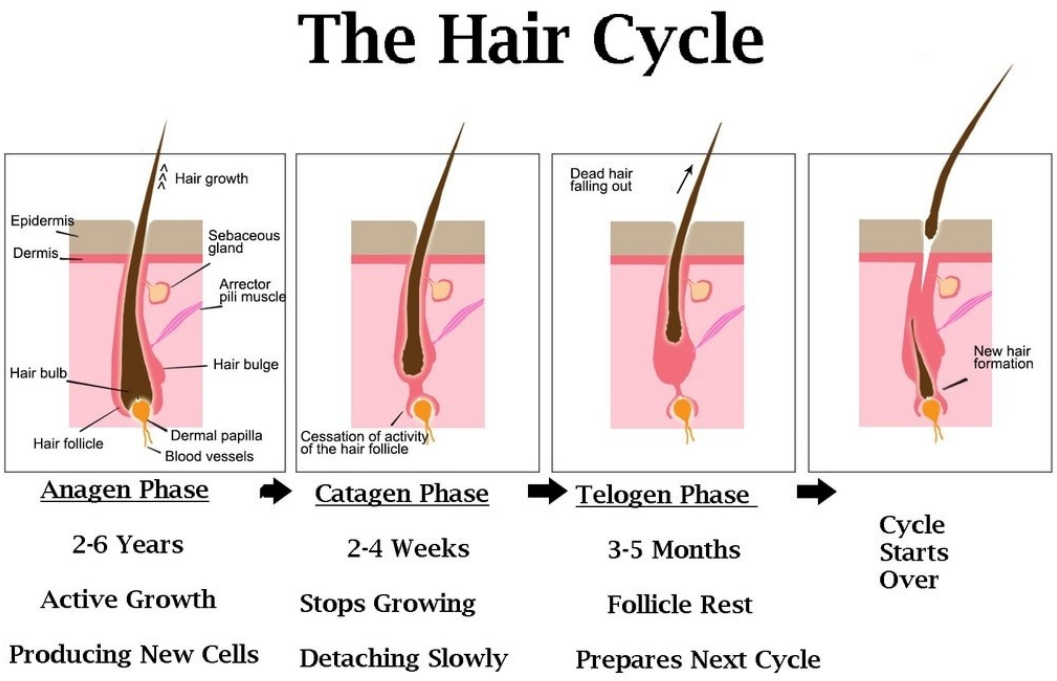


(*The Hair Growth Cycle*, n.d.)

Current treatments for androgenic alopecia

The current treatments for hair loss include:

1. Wigs/ barber's synthetic creations.
2. Surgery –via various implant methods with an unpublished success rates.
3. Minoxidil, Finesteride or alpha-5 reductase blocker medications.
4. Low level laser therapy to increase circulation.

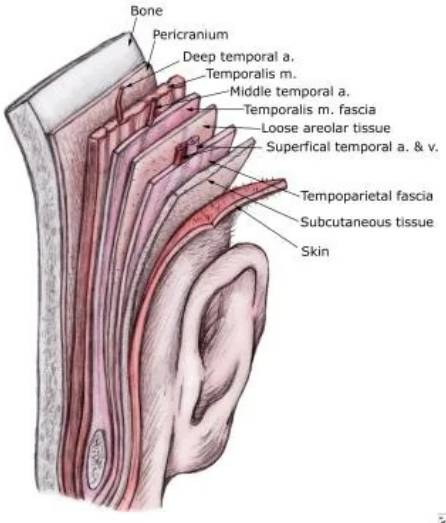
A review of the skull, scalp, cranial blood supply, myology

The human cranium is composed of 29 separate flexible membranous bones, postulated and objectively measured to have movement at the joints or sutures between them. These movements are governed by factors such as muscular contraction, attachments and reciprocal tension membrane forces from inside the skull as well as fascial forces from local and remote locations of the human muscle and skeletal system.

Depending on how they are counted, there are about 30 muscles on each side of the human skull. There are 20 muscles in the neck that directly attach to the skull and the cranio-cervical, superficial and deep fascia, as well as the superficial muscular aponeurotic system (SMAS) (Whitney 2021). The fascial ties are common to the head, neck, and the entire body as one connected unit. Another fascial connection is the aponeurosis that covers the calvarium called the galea aponeurotica; this flat tendon is the insertion of two opposing muscles—the frontalis and occipitalis, also known as the occipitofrontalis. On either side of the skull is a location for the temporalis muscle. Traveling through this vast network of muscles, bones, and fascia are the arteries that supply these structures. The scalp's blood supply arrives via the superficial temporal artery, a branch of the external and internal carotid arteries. The carotid arteries were named by Galen based on the Greek word for stupor, as he found when you compressed them, one could go into a stupor.

The scalp, which tops the calvarium, is a network of adipose tissue, skin, and hair follicles and sits on top of the galea aponeurotica.

The scalp layers from bone calvarium to the skin and hair progress as illustrated below;



Discussion

A novel pathogenisis model for male pattern baldness

The male pattern balding process from start to finish takes approximately 5-25 years to complete. The process is not an acute problem but chronic and progressive for most; stress chronically causes disease, but it is also episodic for most. The hair loss pathogenesis is from terminal hair to vellus to lanugo to bald and then dormancy.

Working backwards from a bald scalp to a healthy scalp with a full understanding of wholism, we see that the end result of hair loss is a dormant or weakly producing hair shaft from a hair follicle that still has stem cells in it.

The "swan song" of a dying hair follicle

The dormant hair follicle once had a miniaturized hair shaft; we called this vellus hair. The hair follicle reacted to elevations of cortisol, and the cortisol caused the hair follicle to be weaker and smaller. This in turn caused a reactive elevation of DHT, which is used by the body to lower cortisol. Lower cortisol levels allow the hair follicle to strengthen or start a new but this does not happen and so elevation of DHT is the swan song of catabolized hair follicles, not the root cause of hair loss-the small hair, now in catagen and doomed, will shed eventually in the exogen phase. Perhaps more rapidly if the levels of cortisol do not decrease. Now the question is, what increased the cortisol?

What triggered catagen – cortisol

We know that elevations of cortisol, either acute or chronic, can throw a hair follicle into catagen leading to hair loss. Stress, shock or trauma do this acutely but usually that happens with the loss of patches of the scalp.

Chronic stress causes the adrenal glands to increase their cortisol output to deal with various types of stress. One such type is structural stress. Structural stresses may present as abnormal cranial bone movement, myofascial dysfunctions and adhesions. Then changes in the shape of bones from years of cranial bone remodeling create a holographic mismatch, leading to increased stress and insidious increases in cortisol, decreases in blood supply and functional hypoxia for the scalp via the superior temporal artery. As this process insidiously happens, silently and slowly, so too does the loss of the terminal hair, propelling it into catagen status (S 2020).

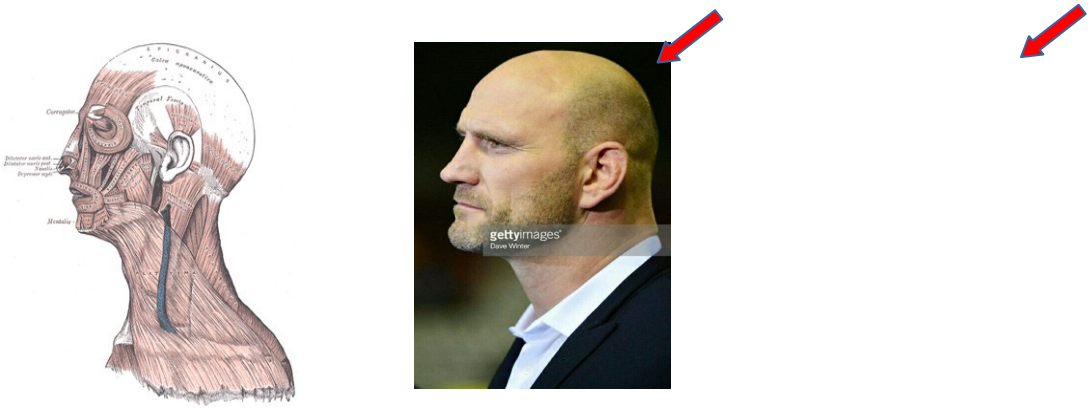
The causes of cortisol excesses are best reserved for future works but includes non-optimal interactions with, structural, chemical, thermal, emotional and geopathic and electromagnetic stressors. Chronic exposure may increase cortisol levels.

Hair follicle miniaturization is complicatedly the product of chronic excess cortisol exposure and slow deprivation of oxygen and nutrients in the case of androgenic alopecia (AA) (Toufexis 2012).

The years of hypoxia also cause muscle dysfunction, which creates fascial distortions that lead to chronic but low force hypertonicity and imbalance; and as Dr. George Goodheart used to say, "Muscles do move bones." And so, Wolf's law takes effect to make the problem chronic. According to this law in part, a bone in a healthy animal will adapt to

the load under which it is placed. Hence, we see actual molding of the skull (Wolff 1892).

So, we start to get growth and shape changes in the cranial bones as a result of persistent and consistent myofascial tension, imbalance, and muscle dysfunction. These osseous changes cause changes in the tension of the galea which cause further fascial distortions and further decreases in blood supply and resultant diminished nutrition to the scalp. As a result, cortisol secretion is stimulated, miniaturization of more follicles occurs, and DHT increases to neutralize the cortisol, and we start over again, year after year; thinner and thinner. The "gravity theory" too has been in play all the while. Except the pulling is the myofascial system, not the gravitational forces of the planet, other wise everyone would be bald. The end product is loss of hair, male and female pattern balding, and distortion of the shape of the cranial bones (Graham Kennedy, 2013). Oddly and overlooked is the fact that the balding of male pattern balding is almost in pattern with galea aponeurotica in many individuals.



The illustration compared to the photo demonstrates galeaic hairloss in the male pattern balding fashion.

Well before the above scenario was probable, cranial imbalances and deviations from normal cranial respiratory movement were an issue. This caused a decrease in blood support through increases in tension and myofascial dysfunction from chronic and acute stress.

These structural stresses that affect cranial movement and scalp mobility and nourishment may occur anywhere in the human body, but most commonly in the neck and shoulders.

<u>Correction</u>
Now that we have identified the pathogenesis. Where do we start on the correction? At the end of course!

In any situation where damage is being done you need to;

1. Get to a safe place – in this case we need to identify areas of cortisol stimulation and start addressing them. This may be very individual. (i.e. overwork, overtraining, emotional situations unhandled, etc.)
2. Stop the damage – The hair follicle has been deprived of vital blood, oxygen and nutrients. It may be using anaerobic glycolysis to survive. The end product is lactic acid. Some people notice this because of soreness, itching and burning. That means that NAD is being used for this process so testing for Niacin not niacinamide is a first step to stopping damage.
3. Next to control damage we must discourage the local testosterone conversion by 5 alpha reductases. We can use frequently applied essential oils and topicals for this.
4. Check each of the 29 bones of the cranium for holographic subluxations.
5. Check for cranial faults and sutural faults.
6. Release the frontooccipitalis muscles.
7. Evaluate all muscles of the skull for proprioceptive dysfunction.
8. Evaluate the entire stomatagnathic system.
9. Evaluate fascial balance and for adhesions using the midline scalp pull test.
10. General fascial release per fulford may be achieved by using percussion at the following locations:
    - left and right deltoid tuborosities
    - left fibular head, C2, L2 and S2
11. Evaluate and release the sacrum and coccyx.

Now that you have addressed the various structural concerns all of the following nutrient areas need to be assessed and corrected:

1. Scalp cortisol challenge
2. Zinc status
3. B3
4. Methylation status – homocysteine protocol (Schmidt)
5. Essential fatty acid blood spot profile
6. Vital amino acids needs must be assessed and met.
7. Cranial remolding exercises must be performed three times daily. Cranial molding exercise may cause cranial faults so the cranium should be re-evaluated to ensure no harm has been done. These exercises must be prescribed based on the holographic imbalance in each cranial bone identified (e.g. parietal bulge is most common)
8. Calvarial muscles must be evaluated for any types of dysfunction
9. Each muscle of the facial skull muscle be evaluated for dysfunction
10. Topical and internal use herbs that improve blood supply are adjunctive and needed.
11. Adrenal function must be assessed and addressed per the AK Synopsis.

Conclusion

Thirty-five years prior to this paper, a discovery was made that described, not the cause but the end result of one process of hair loss. Dihydrotesterone elevations are the end

result and hair unit “swan song” from years of blood deprivation and all of the life-embodying factors that it carries to the tissues and cells of the scalp. Applied kinesiology allows the pieces of this puzzle to be found and arranged in a fashion that allows us to see the big picture of the puzzle completed. To restore full normal function to the hair unit requires a panopoly of factors for restoration and stabilization of a weak and ailing hair units and the scalp they reside in. But this can be done by those willing to put forth a little work and effort.

Acknowledgments are made to Harmonic Energetics, Integrated Healthcare of Montclair LLC, and the ICAK.

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