What Is Delayed Growth?
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The asynchronous cyclical nature of human follicular activity contrasts sharply with the synchronized "molting" pattern seen in other mammalian species. In the process of molting, animals shed their hair in a synchronized, seasonal pattern to adapt to the changing climate. This shedding of hair is controlled by mediators released by the hypothalamus, pineal, and anterior pituitary.

Humans differ from other mammals in that their growth cycle is almost totally asynchronous, and this pattern is established shortly after birth. Human hair has a growth cycle of approximately three years. Since human hair grows approximately 1/2 inch per month, it would reach a length of about 18 inches before it is shed. Therefore, in the average person, hair length is limited to approximately 1-1/2 feet. There is, of course, significant normal variation with hair cycles varying in each individual and from person to person. At the extreme end of the bell curve, continuous patterns of growth may be seen (Angora). In a given period of time, about 13% of hair is in the resting (telogen) stage, which lasts about three to four months. For example, if the average person had 100,000 hairs on his head, approximately 90 should be shed each day, and 90 would begin to re-grow.

Generally, following a transplant, all of the transplanted hair is shed in an anagen effluvium and enters a resting phase. If all the hair was to simultaneously begin a new anagen cycle, the asynchronous normal growth pattern in man would be effectively converted to the synchronous growing/molting pattern of animals, and much of the new hair would fall out during the next normal telogen three years later. This obviously doesn’t happen to any significant degree.

To prevent this from happening, and to keep our hair cycle “human,” there are at least two possible mechanisms at work. In the first, each hair might resume its growth cycle at the point where it was interrupted, thus continuing the previous totally asynchronous pattern of growth. In the second possibility, each hair would begin a totally new three-year cycle. In this case, if all the hair was to begin growing simultaneously, this new hair would fall out at approximately the same time three years later. In the second scenario, in order for nature to restore its normal asynchronous pattern, it would have to delay the onset of growth in a portion of the follicles. In theory, to be totally synchronous, new crops of transplanted hair would continue to appear over an extended period of time, possible up to three years.

It is unclear which of these two physiologic mechanisms best explains the natural course of events, but it is likely that both are at work. In the first, abundant new hair growth would be seen at three to four months with each follicle resuming its original cycle. In the second scenario, hair would gradually reappear over an extended period of time with each follicle at the beginning of a full new cycle. It is noted that in some patients, substantial hair growth is delayed significantly past the usual “three to four months,” with some new growth occurring as late as a year or more after surgery. It is possible that in this subset of patients, the second natural mechanism is at work.

We wonder if this phenomena of delayed growth is unique to the newer techniques or has always been present and is only now being recognized. With 10 mm grafts, containing an average of 20 hairs, a delay in the growth of some of the hair would largely go unnoticed. With the use of smaller grafts, such as minigrafts, but especially with micrografts and follicular implants, any delay would be immediately apparent. In fact, it has been the experience of these authors that the appearance of the large grafts often worsens with time as the full complement of hairs in each graft become visible.

If delayed growth is indeed a new phenomena unique to the implantation of very small grafts, it is possible that the growth cycle of the hair in these grafts may react differently to the trauma of transplantation than those in the larger grafts. In other words, the desiccation, temperature change, and mechanical trauma to which small grafts might be more subject actually cause a delay in growth in situations where there was not quite enough injury to completely destroy the follicle. Thus, small degrees of trauma may either induce the second physiologic mechanism to occur, or may cause a more pathologic delay in growth. Of course, with increasing trauma, poor growth or no growth would be the result and this “point of no return” must be elucidated in carefully controlled studies. An additional type of trauma that may contribute to delayed growth has been recently reported by Drs. Cooley and Vogel. They suggest that when the dermal papillae is lost during graft preparation and handling, it must regenerate from the fibrous root sheath before the hair will regrow, and this regeneration can often take several months. The role that this important mechanism plays in the delay that we see clinically must also be determined.

Our guess is that “delayed” growth has always been part of the transplant process, representing a normal physiologic shift in the growth cycle on one hand, and a reaction to sublethal injury on the other; with the very small grafts making both of these changes more obvious. What percentage each represents and how much of “delayed growth” will turn out to be no growth at all still needs to be determined. But before we panic about “perceived no growth” and before we set our patients up for unrealistic expectations about how soon their growth may occur, we should work to have a better understanding of all the biologic factors that impact our surgery.

References