VIP INTERVIEW
Birgit Thilander

Edited by: Samir E. Bishara, BDS, DDS, DOrtho, MS

Birgit Thilander, LDS, Odont Dr (PhD) was professor and chair of the Department of Orthodontics, University of Umeå (1963 to 1968), and the University of Göteborg (1969 to 1991), and visiting professor at the National University of Colombia, 1993 to 1999. She received Honorary Doctorate degrees from the University of Helsinki, Finland; University of Bergen, Norway; and the National University of Colombia in Bogota. She is an Honorary Member of many national and international societies, including the World Federation of Orthodontists and the European Orthodontic Society. Dr Thilander is the recipient of 15 distinguished awards and was the president of the European Orthodontic Society in 1981. She has been an invited speaker in most countries in Europe, in the United States, Canada, South America, Japan, China, Hong Kong, Korea, and New Zealand. In addition, Dr Thilander is the author of 3 textbooks and the editor of another textbook. She has published more than 180 articles in international journals and 15 chapters in different textbooks.

It might be of interest to start this interview by asking about some of the highlights of your long and distinguished career. Let us start by finding out from you a little about graduate orthodontic education in Sweden.

To be a specialist in Orthodontics (LDS) in Sweden, you need to have experience of at least 2 years in general practice to be accepted for a 3.5-year full-time postgraduate program. These programs are given at 10 different institutions in the country. It is very difficult to be accepted as a postgraduate student, which will explain why only 10 to 15 new orthodontists are examined each year. In all of Sweden, there are 250 specialists, most of them employed in the municipal dental service, which offers orthodontic treatment free of change for children and adolescents. Very few orthodontists are in private practice.

The research education is given at the 4 universities during a 4-year full-time period with compulsory courses in scientific knowledge and special courses related to the aim of the research project. The studies end by completing a thesis that has to be publicly defended. Then you become an Odont Dr (equivalent to PhD). Some young students prefer to combine these 2 programs, which means a full-time program of 6 to 7 years.

What is your philosophy of applying the results of animal studies to clinical orthodontics?

The question in each of my projects was originally raised in the clinic. Due to ethical reasons, I had to find an experimental model (rat, guinea-pig, rabbit, beagle dog, monkey, and pig) to get the answer to my clinical problem. The results from the experiment then had to be tested in the clinic. Only after a follow-up period of at least 3 years, I would consider that we have acquired enough experience to give recommendations to clinicians. However, I prefer to follow my patients for a longer period, which has resulted in important information, eg, those obtained from the 10-year follow-up study of implant placement.[1]

In which area of research did you start your academic career?

My interest in research started early with problems related to the temporomandibular joint. After a 3-month visit to Professor Petrik in Vienna, which was originally aimed to learn more about activator treatment, I started the anatomical-histological studies of the condyle, temporal component, discus, insertions of muscles (including fiber analysis), and the capsule. The innervation of the capsule was followed by some neurophysiological studies on pain (free nerve-endings) and jaw mobility (complicated nerve-endings/proprioceptors). All this became the topic of my thesis. It might be of interest to note that I defended my thesis the day after my husband (an oral surgeon) had defended his own thesis. This was considered a special event in the Swedish dental world, a huge accomplishment for a couple with 2 small children.

So what came next on your research "menu"?

The studies on the TMJ region[2] were followed by a series of studies on the cranial base,[3] nasal septum,[4] and facial sutures.[5] Once upon a time, we had learned that all these structures were "active growth centers". During a long period, I was occupied by charting these structures using different methods, with the aim to find an answer to the mechanism behind dentofacial development and growth. All studies proved that these structures are passive in nature.[6] As adaptive zones, the TMJ region and the sutures thus can be influenced by orthodontic/jaw-orthopedic treatment. This hypothesis on the association of function and growth was tested in clinical studies, eg, treatment of crossbite with maxillary expansion,[7] Angle Class II cases with different functional appliances,[8] and Class III cases with chin-cap.[9]

Long-term follow-ups indeed have stressed the importance of differential diagnosis. Today, all of us know that the jaw relationship is the result of the growth in the nasomaxillary complex and the mandible, together with the mandibular rotation (upward or downward), which is of importance for the treatment

planning. However, regarding the transverse dimension, the differential diagnosis is often neglected, eg, in patients with crossbite. Our follow-up study of unilateral crossbites[10] clearly could show that maxillary expansion resulted in relapse in those cases with a narrow maxillary arch width combined with a broad mandibular width at the crossbite side.

Thus, "growth" and "function" have been the central interest during my whole life as a researcher. And the discussion on temporomandibular dysfunction and malocclusion is of current interest to me. We need to find out whether there is an association and, if so, in which cases.

In an earlier visit, we discussed that we both have been involved in longitudinal cephalometric studies. Would you share with the readers the basis for the Swedish study and some of the results obtained?

A longitudinal cephalometric study of Swedish males and females with "ideal" occlusion (not orthodontically treated) followed from 5 to 31 years of age[11] has verified that ethnic differences in facial traits exist, which you also have shown in many of your own publications. Thus, cephalometric standards of ethnic groups are important in orthodontic diagnosis and ought to be subject of an Atlas.

The study also verified that facial pattern changes during the whole observation period. The increase in length of the mandible, together with its upward rotation, resulted in a change of the facial convexity—from slight convex to straight, and even to slight concave profile. The soft tissue profile also changed with age. The most notable observations were the prominence of the nose in males and the decrease in thickness of the upper lip, already from 13 years in females.

In regard to the dental relationships, the incisors continuously achieved a more proclined position up to the 16-year recordings, while great individual variations were observed in both the young adult and adult periods. Furthermore, a biometric analysis on the study casts has shown lack of stability of the arch form in persons with normal occlusions when passing from adolescence into adulthood, often resulting in crowded mandibular incisors.[12]

The results from the "ideal" subjects will bring the matter of "orthodontic relapse versus natural development" to a head. Additional support for this comment is the experiences from the 10-year follow-up of implants in adolescents, which showed that teeth adjacent to an implant continuously erupt (0.1 mm/year), even in some adults.[1] Those 2 longitudinal studies clearly will verify the importance of our knowledge of dentoalveolar development.[13]

Still, we do not know the mechanism behind tooth eruption, so we cannot predict when it will stop. At this point, a follow-up study of patients with implant placement in adult ages is in progress.

From looking at your publications, I realize that you have done significant research in the area of periodontics as it relates to orthodontic and non-orthodontic tooth movement. Please summarize for us some of your research in that area, as well as the highlight of the findings.

In the 1970s, much time was focused on patients with periodontitis. Some patients were unhappy with anterior spacing resulting from the pathological migration of the teeth and wanted the spaces closed orthodontically. In those days, we did not know the effect of tooth movements in such patients. My question to the periodontists was: Will orthodontic forces result in further marginal bone loss? Since they were as ignorant as I was, we started an experimental study in Beagle dogs with the aim to find an answer. The experimental findings told us that orthodontic tooth movements could shift a supragingivally located plaque into a subgingival position, causing infrabony pockets, in contrast to movements in plaque-free regions. We had a long series of different studies in dogs and monkeys aimed at studying tissue reaction of different tooth movements, the effect of fibrotomy (relapse or not), bone regeneration in orthodontically produced dehiscence, tooth movement in reduced alveolar bone height, and tooth movements through the midfacial suture.

All these studies stressed the importance of adequate plaque control during the orthodontic treatment period, which has to be performed with light forces and good anchorage. Bodily movement is preferable to tipping, and intrusion indeed needs caution. No significant difference existed between fibrotomy performed prior to or after the orthodontic tooth movement, in regard to the prevention of relapse. Alveolar bone loss (dehiscence and fenestration) can be produced in the alveolar plate by tooth movements, but the bone will be reformed when the tooth is moved back again to its original place. The volume (thickness) of the covering soft tissue must be considered as a factor which may influence the development of gingival recession following bone loss. A tooth with normal periodontal support can be orthodontically moved into an area of reduced alveolar height while maintaining the height of the connective tissue attachment level and alveolar bone support, provided that very light forces are used. A tooth can pass the suture area if the suture is closed (though with extensive root resorption); this is in contrast to an open suture, in which the suture will be dislocated in front of the tooth. Most of those results are presented in a separate chapter on tissue reactions in orthodontics.[14]

All these experimental studies were followed by treatment of patients, both periodontic patients and those with a need for orthodontic-prosthodontic or orthodontic- surgical treatment. I must confess that I have treated lots of adults with long follow-up periods before giving recommendations to the general clinician. It has been of great value for me to have the privilege of working together with many serious specialists from other fields, as well as with skilled general practitioners. And my dear husband Holger (deceased some years ago) was my real supporter in scientific and social life.

You have had an illustrious and lengthy career in orthodontics. You have seen enormous changes in our profession in the last 30 years or so. Where do you see our specialty heading both short-term and long-term? And what advice do you have for the younger generation of orthodontists?

I am afraid that the postgraduate students, as well as many "experts" in orthodontics, are more interested in brackets and wires, bonding materials, and mini-implants as anchorage units than in basic knowledge. Every orthodontic congress offers an exhibition area filled with orthodontists gathered around hundreds of stands and buying these materials, recommended by the different manufacturers. Of course, we need these facilities, but we must be aware of the tissue response and the probable risk of tissue damage. The lecture rooms with clinical recommendations (often without serious background) are well frequented, in contrast to those rooms announcing a scientific issue of basic character.

So, I think that we have to spend more time in basic knowledge on dentofacial development and growth. This is of importance for diagnosis/differential-diagnosis. We need to explore function and tissue reaction (including adverse effects) caused by biomechanical parameters (force, type of tooth movement, treatment duration). How to explain to patients the difference between relapse versus natural development? I am convinced that such in-depth teaching during the postgraduate period will encourage some young people to get involved in orthodontic research, both clinical as well as experimental, and with that approach, bring more knowledge to our special field.

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