

PULP VESSELS: A FACTOID

This **FACTOID** discusses the morphology & physiology of vital pulp vessels. Until histologists of the 1800's could prepare sections of the tooth & pulp—knowledge of the pulp was limited & gross anatomists were quick to discredit the microscope.

Most new ideas & those who support them often find others who oppose them since it represents a rethinking of previously accepted knowledge. Von Leeuwenhoek's microscope opened a new world of observation—creating a technology that remains in a dynamic state of evolution. In 1844, Schleiden proposed his vital cell theory—in just 100-years we can now dissect components of the atom.

On April 3rd 1889, Dr. Carl Heitzmann gave an oral presentation on “The Future of Microscopy” to the Brooklyn Medical Microscopical Society of New York. He discussed the histological advances that had been made since the mid-1800's from research laboratories in Europe. However, there remained a “dark-side” that Heitzmann & his colleagues had suffered from adverse comments by individuals who had no idea or value of microscopy. Heitzmann commented that he had “exhausted his forces in swimming against the current of vulgarity & superficiality” he faced in Europe. So strong were his antagonists that he was denied an academic position at Vienna. Consequently, he came to the United States & opened a private research laboratory on Nov 1st 1874 on the top floor of his rented house—unable to speak English with any strong capacity. After 15-years of many challenges, he could finally boast many 15-tables & 15-microscopes for clinicians who wished to learn histological & microscopic techniques at his research facility. Heitzmann's philosophy was **“To try to solve Nature's puzzles is an occupation worthy of an honest man and a man fond of thinking. . .Nature will never disappoint us if we seek the truth. . . Ingratitude, vulgarity and profanity do not exist in the laboratory; here we are the high-priests of the virgin science”**. In a moment of reflection, Heitzmann honored his “only true friend I ever had. . .a most generous and magnanimous man I have ever met.” He recognized C. F. Woerishoffer as his teacher of a discipline & his supporter to the new era of scientific work—we must also know his name.

Our profession owes a great deal to Dr. Heitzmann—the first histologist to successfully use Canada balsam as a mounting medium that provided intimate visualization of pulp tissues that had before suffered little investigation.

In 1840, Richard Owen published his monograph “Odontography” in which he discussed the comparative anatomy of teeth. Owen described the dental pulp as “consisting of semi-opaque polyhedral granules or cells suspended in a clear *matrix*, and the whole is inclosed in a tough transparent membrane which forms the outer surface of the pulp”. His colleague Cuvier described “the free surface of the granular [pulp] tissue as covered by a particularly dense, structureless-pellucid membrane termed the ‘*preformative membrane*’ from which dentine commences. Once dentine is deposited, the blood-vessels soon penetrate the granular pulp”.

In 1897, Lepkowski showed slides of small branches that had penetrated from the inferior alveolar artery that entered the dental papilla. As the papilla matures through various stages to the mature organ, the vessels become as a Lombardy poplar tree that is devoid of its leaves. The few central arterioles increase in diameter whereas the muscular lumen wall is reduced in thickness with many branches projecting to the periphery that terminates in a dense zone of small subodontoblastic capillaries that form an extensive network amongst the odontoblasts—being most dense in the coronal & pulp horn. Small open pores (fenestrations) are occasionally observed that provide for the rapid transport of nutrients during active dentinogenesis. These terminal capillary-arterioles proceed directly into small venules with very thin walls, presenting a larger lumen than arterioles—efferent flow follows a reverse pathway emptying blood from the pulp.

At the 1984 Pulp Biology Conference, Dr. Takahashi presented his unique 3-dimensional SEM analysis of the normal vital pulp. He showed detailed vascular resin casts of pulps in various stages of growth & different stages of inflammation by injecting a low-viscosity resin directly into the vital blood vessels of an anesthetized dog. Following removal of the mineral & organic tissues, he observed the remaining resin casts by SEM. His unique technique provided a new dimension in the differentiation of pulp arterioles from venules. He was first to show a venous-venous

anastomosis (VVA) as well as a “U-turn loop”, which we now understand to provide a unique role in regulation of blood flow. At that same Conference, Dr. Karen Heyerass-Tonder differentiated between the microvascular (MV)-part of the *Starling equation* (Pi) & interstitial tissue pressure (TPi). Her unique micropuncture techniques allowed for the direct measurement of blood pressure in different vessel segments, e.g. arterioles, capillaries & venules as well as to differentiate extravascular tissue pressure. Dr. Tonder demonstrated a wide variance of pressure values by the TPi that was due to the **low compliance** tissue pressure of the pulp. The vital human pulp is a unique vascular tissue of **LOW COMPLIANCE** being rigidly bound within the un-yielding dentine and enamel—the pulp simply has no space in which to expand during any sort of stimulation—whereas our vital external skin—if pinched--is **highly compliant** since it easily expands without any constraint.

All pulp vessels—arteries & veins—enter & exit through numerous foramina of the root apex that is often surrounded by root cementum—especially as the tooth ages. These vessels project from the rich vascular vessels that feed into the alveolar bone & then into the periodontal ligament space of richly filled cells, collagen & oxytalan fibers.

Dr. Takahashi demonstrated that arteries of the pulp have unique arteriole-venous shunts that can rapidly open & re-route arterial blood back into large veins & out through the apex, removing the vascular pressure-load on the more peripheral vessels of the odontoblastic cell layer & consequently, the physiology that Dr K. H. Tonder demonstrated is an explanation of why the dental pulp does not actually “strangle itself” as a result of local inflammation. Data of Stenvik reported tissue pressure measurements of up to 50-60 mm Hg, Whereas, simultaneous measurements from inflamed & normal pulps made by the physiological research of van Hassel, Tonder & Kvinsland have shown that increased pressure may be a local phenomenon, which is not conveyed to the rest of the pulp.

In skeletal muscle, the local control of the arteriolar vessel diameter will greatly influence blood flow, whereas in the vital pulp, only a small part of the total pressure fall is located in the precapillary micro vessels. Thus, local control of the vessel diameter in the pulp arterioles would not significantly affect pulp blood flow.

Tonder described the vascular build-up as a so-called “vicious circle” during pulp inflammation. She described the inflammatory-induced vasodilatation & increased capillary permeability results in an augmented net fluid filtration from the blood vessels & into the tissue, which in turn causes a steadily increase in local tissue pressure. Gradually as the pressure on the outside of the blood vessels (tissue pressure) rises, the venous vessels may be strangled, resulting in an increased venous vascular resistance, thus causing stagnation of the blood circulation with resultant ischemia & necrosis.

CLINICAL CONSIDERATIONS

Under normal operative / restorative procedures, the vessels of the pulp can easily accommodate their physiology to various stimuli: bur-drilling & vibration, heat from exothermic setting reactions of cements, liners, self curing adhesive systems as well as intense transfer of heat of polymerization from lasers & other heat generating devices as well as to miss-placed air-tip direction onto prepared dentine & of the pathological (non-clinical) insult of caries (acute or chronic).

It is important you know that Dr. Henry Van Hassel demonstrated that the dental pulp does not die by immediate **strangulation**—the pulp suffers episodic bouts of **compartmentalization**—meaning unless there is an immediate iatrogenic insult to the vital pulp, most have a great **inherent healing capacity**—only die in compartments.

Being the human bodies most unique **low-compliance tissue**—soft tissue encased in a rigid dense substrate of dentine & enamel—perhaps the biggest physiological challenge the clinician faces is when they create an accidental (**iatrogenic**) pulp exposure that destroys local pulp cell stroma, nerves & vessels.

The most important clinical treatment is to prevent all bacterial & operative debris from entering the pulp & then to isolate the exposure & provide a biologically compatible antiseptic treatment & to provide proper **haemorrhage** control before placing a **complete bacteriometric restorative seal**.

To understand the proper treatment of an exposed vital dental pulp, please read the following FACTOID that covers HÆMORRHAGE CONTROL.