

# Optical Coherence Tomography for Ophthalmic Imaging

**Akash K Singh, PhD**

IBM Corporation Sacramento, USA

## Abstract

Understanding the etiology and evolution of the vulnerable coronary plaque is important for the early detection, treatment, and prevention of coronary artery disease. Intravascular optical coherence tomography (OCT) enables imaging of the coronary arteries in vivo with sufficient resolution to accurately differentiate arterial pathology, however, the clinical utility of this technology has been limited due to slow image acquisition rates. The development of high-speed Fourier-domain OCT techniques, including optical frequency-domain imaging, enables comprehensive microstructural imaging of long coronary artery segments. Other OCT advancements, including polarization sensitive OCT provides complementary birefringence information that is related to tissue composition. Together with new image processing, acquisition, and display techniques, these advances have enhanced the usability and utility of intracoronary OCT, bringing it closer to becoming a mainstream imaging modality in interventional cardiology.

**Keywords-** Cardiovascular, coronary arteries, intravascular imaging, optical coherence tomography (OCT), optical frequencydomain imaging (OFDI), polarization sensitive optical coherence tomography (PS-OCT).

## I. INTRODUCTION

The human eye occupies a unique place in clinical medicine. Physiologically, the eye represents the most complex sensory input to the central nervous system. The significance of visual information means that ocular disease and visual function impairment can have a devastating impact on a patient's quality of life. Structurally, the unique and specialized anatomy of the eye provides a level of accessibility to internal structure not available in any other organ in the body. Both the anterior and posterior chambers of the eye are amenable to direct visual observation, facilitating the diagnosis of ocular pathology. The uninterrupted optic pathway to the posterior eye allows non-invasive inspection of nervous and vascular tissue, making the eye an important indicator of not only ophthalmic but also systemic vascular and neurologic disease. A basic ophthalmic exam relies on standardized visual function tests and slitlamp observation to assess visual integrity and to evaluate ocular manifestations of systemic disease. Current clinical evaluation often rests on direct and sometimes subjective

visualization of ocular anatomy and reduced performance on visual function tests. In some cases, however, observable loss of visual function only occurs after irreversible microscopic changes in ocular anatomy. Micron scale high resolution tomographic imaging of the anterior and posterior eye would be a powerful diagnostic in these cases, by providing quantitative evaluation of ocular microstructure and the potential for early diagnosis and treatment of disease before visual function loss. In the anterior segment, high resolution imaging would permit the evaluation of pathologies of the cornea, iris, lens, and anterior chamber angle. Accurate, non-invasive measurements of corneal curvature and thickness would also be useful in treatments where precise biometry is necessary, such contact lens fitting, intraocular lens implant power calculation, and real-time monitoring of corneal refractive surgery. In the posterior segment, tomographic imaging of the retina and optic nerve head would allow quantitative evaluation and early treatment of degenerative retinal diseases such as open-angle glaucoma, macular degeneration, and macular edema. Several techniques for ocular imaging are currently being used or investigated for implementation in clinical practice. Fundus photography is routinely performed to document changes in optic disk appearance associated with glaucoma [11]. Fluorescein angiography, which involves systemic intravenous injection of fluorescein dye prior to fundus photography, is able to delineate vascular alterations such as aneurysm, neovascularization, ischemia, occlusion, hemorrhage, and edema [11]. A and B scan ultrasonography are currently employed in the evaluation of intraocular masses and tumors, and the measurement of axial eye length. In typical clinical instruments, however, the depth resolution of ultrasound is limited by the wavelength of sound in ocular tissue to about 150  $\mu\text{m}$  [2,3]. Recent advances in high-frequency ultrasound have reduced the minimum resolution to about 20  $\mu\text{m}$ , but with a penetration depth limited to the first 4 mm of the anterior segment [4]. Ultrasonic imaging also requires either physical contact between the eye and the ultrasonic transducer or saline immersion of the eye. X-ray computed tomography (CT) and magnetic resonance imaging (MRI) are useful in the evaluation of ocular neoplasms, inflammatory masses, orbital fractures, and foreign body detection. Like ultrasound, both imaging techniques are important in a variety of clinical settings; however, their resolution is currently limited to hundreds of

microns [1], SI. By applying laser confocal imaging to the eye, scanning laser ophthalmoscopy (SLO) [6] and scanning laser tomography (SLT) [7] can accurately profile changes in the contour of the fundus surface and quantify cupping of the optic disk with micron scale lateral resolution. However, direct depth resolved tomographic imaging of the fundus is limited to about 300 pm accuracy by ocular aberrations and the numerical aperture available through the pupil. Indirect thickness measurements of the birefringent retinal nerve fiber layer may be obtained by coupling a Fourier ellipsometer to an SLO in order to also measure the polarization state of reflected light [8]. We have developed a new technique for micron scale resolution cross-sectional imaging of ocular and other biological tissue, called optical coherence tomography (OCT) [9,10]. OCT is similar to Bscan ultrasonic imaging, except that image contrast relies on differences in optical rather than acoustic backscattering characteristics of tissue. In contrast to ultrasound and nonlinear optical gating techniques, low-coherence interferometry is used to resolve the position of reflective or optical backscattering sites within a sample. Two-dimensional tomographic images of a thin, optical slice of tissue may be obtained with 10  $\mu\text{m}$  longitudinal and lateral resolution. Optical heterodyne detection and the application of noise-reduction techniques originally developed for optical communication achieve a sensitivity to reflected light as small as 10<sup>-8</sup> of the incident optical power. OCT is non-contact, non-invasive, and has superior resolution to conventional clinical ultrasound. Unlike SLO and SLT, the optical sectioning capability of OCT is not limited by the pupil aperture and ocular aberrations. OCT may be implemented in a compact, low-cost, fiber-optic based interferometer that is easily coupled to existing ophthalmic instrumentation. In this article, we demonstrate high-speed in vivo OCT imaging in both the anterior and posterior eye, and highlight the system's potential

usefulness for the early diagnosis and quantitative monitoring of a variety of ocular diseases and treatments.

## II. OPTICAL COHERENCE TOMOGRAPHY

OCT is a two-dimensional extension of optical coherence-domain reflectometry (OCDR), a technique originally developed for high resolution and high sensitivity characterization of optical waveguides and fiber components [11-14]. More recently, low-coherence reflectometry has been applied to single axis profiling of optical reflectivity versus depth in a variety of biological tissues [15-20]. We have implemented a high-speed fiber-optic low-coherence interferometer that forms the basis of the OCT scanner [10]. As depicted in Fig. 1, broad bandwidth, lowcoherence light from a superluminescent diode (SLD) source is coupled into

a fiberoptic Michelson interferometer. The 843 nm light is divided at a 50/50 fiber beamsplitter into reference and sample paths. Light retroreflected from a variable distance reference mirror is recombined in the beamsplitter with light backscattered from the patient's eye. Coherent interference between the reference and sample beams is detected by a silicon photodiode, followed by signal processing electronics and computer data acquisition. The temporal coherence of the light source determines the resolvability of backscattering or reflective sites located at different depths within the eye. A coherent interference signal is evident at the detector only when the reference arm distance matches the optical length of a reflective path through the eye to within the source coherence length, typically 10-20  $\mu\text{m}$ . A longitudinal profile of reflectivity versus depth into the sample is obtained by rapidly translating the reference arm mirror and synchronously recording the magnitude of the resulting interference signal. Since the scanning reference mirror traces out the convolution of the source field autocorrelation function with the sample reflectivity profile, the minimum depth resolution depends entirely on the source spectral bandwidth and not on the available numerical aperture, as in confocal imaging systems such as SLO and SLT. Assuming a gaussian lineshape, the fullwidth-half-maximum (FWHM) autocorrelation width or ranging resolution  $\Delta L$ , may be calculated from the inverse Fourier transform of the source spectrum  $\Delta L \Delta \lambda = \lambda^2$ , where  $\lambda$  is the source wavelength and  $\Delta \lambda$  is its FWHM spectral bandwidth. The current OCT scanner employs an SLD source with a bandwidth of 30 nm, leading to a measured ranging resolution of 14  $\mu\text{m}$  FWHM. The SLD is advantageous compared to other low-coherence sources, since it is compact, low-cost, and emits much of its light into a single spatial mode. Analogous to the ultrasound B mode, two-dimensional images of optical backscattering from within the eye are created from a sequence of single, uniaxial OCDR scans. As depicted in Fig. 2, crosssectional OCT images are obtained by repetitively translating the reference mirror while scanning the probe beam across fluctuations in signal power are dominated by the quantum statistics of light returning from the reference arm. The signal-to-noise ratio (SNR) in this optical shot noise limit determines the minimum detectable reflectivity in the eye, and can be mathematically described by: where  $\eta$  is the detector quantum efficiency,  $h\nu$  is the photon energy,  $\Delta \nu$  is the noise-equivalent bandwidth of the demodulation filter, and  $P_s$  is the power incident on the sample. The sensitivity to weakly reflected light depends only on the detection filter bandwidth and the available optical power, which is ultimately limited by tissue damage thresholds. The filter bandwidth is chosen wide enough to accommodate the maximum signal bandwidth  $\Delta \lambda$  where: Equations 1-3 demonstrate that

detection sensitivity, or SNR, trades-off linearly with both depth resolution and scanning velocity. To facilitate real-time in vivo imaging, a reference mirror scanning velocity of  $v = 156 \text{ m/s}$  is employed in the current system, leading to a doppler shift frequency of  $f \sim -400 \text{ kHz}$ . With a detection bandwidth of about 40 kHz and 175 pW incident power on the eye, a sensitivity to reflected light smaller than 50 femtowatts is obtained (95 dB equivalent SNR). Fast image acquisition is especially important to minimize motion induced image degradation for in vivo ocular tomography. The total image acquisition time is given by: where  $L$  is the axial scan length,  $n$  is the number of transverse pixels in each tomograph, and  $g$  is the reference mirror translator duty cycle ( $g \approx 0.8$ ). In our current system, a typical 100 transverse pixel retinal tomograph with a scan depth of 3 mm may be obtained in about 2.4 s. A typical tomograph of the full anterior chamber containing 200 axial 8 mm length scans requires 12.8 s. Proportionately faster image acquisition times may be obtained by either reducing the number of transverse pixels, or by linearly increasing both the scanning velocity and incident optical power according to Eqs. 2 and 3. The current incident power of 175 mW meets a conservative interpretation of the ANSI exposure standard, which limits the power for permanent ocular exposure with a full pupil aperture and a non-scanning probe beam to 200 pW at 830 nm [21]. This extended viewing limit is raised to 1.3 mW if one accounts for the fact that the OCT system employs a relatively narrow scanning probe beam. Thus, future increases in incident power should allow markedly increased scanning velocities and corresponding sub-second image acquisition times.

### III. in vivo imaging of the retina

The high resolution and high sensitivity of OCT in the posterior eye makes it uniquely suited among existing ophthalmic imaging techniques for clinically relevant tomography of the human retina. Micron scale anatomic changes in the retina are potentially pathognomonic, limiting the applicability of comparatively low resolution imaging techniques such as CT, MRI, and conventional ultrasound for nuclear layers, respectively, which both consist of neuron cell bodies and appear as unlabeled darker layers indicative of low scattering. The bright red layer below the retina corresponds to the choriocapillark, a dense interconnected network of small, highly scattering blood vessels. The retinal image acquisition time of 2.4 s is too slow to completely eliminate image artifacts due to patient eye motion during scanning. To compensate, the tomography was digitally processed to remove artifacts due to involuntary motion. The original uncorrected tomograph, shows that ocular motion causes small oscillations in the contour of the nominally flat macular region. The image artifacts

due to scan-to-scan axial variation may be corrected by a cross-correlation scan re-registration technique. An estimate of the axial ocular motion is obtained by selecting the longitudinal indices, which correspond to the locations of the peak values of the crosscorrelation between adjacent axial scans. The peak indices, interpreted as the actual retinal contour corrupted by motion artifact, are then digitally low-pass filtered to evaluating diseases such as glaucoma, macular edema, and macular degeneration. The available numerical aperture and ocular aberration limit the resolution at the retina of scanning confocal imaging devices, while acoustic attenuation makes remove the presumably artifactual high spatial frequency variations in axial position. A comparison of the original noisy and filtered contours permits re-registration of shifted scans to form an uncorrupted image. The posterior segment unreachable by high frequency ultrasound. 100 (horizontal) x 250 (vertical) pixel OCT tomograph obtained from the macular region of a volunteer. The foveal region of the macula, specialized for maximum visual acuity, is visible as a characteristic thinning of the retina. At the foveal depression, most of the retina anterior to the photoreceptor layer (PRL) is displaced laterally to allow free passage of light and to maximize the concentration of cones in the area of central vision. The photoreceptor layer, which consists of the photoactive segments of the retinal rods and cones, appears to be minimally backscattering in the tomograph. Alternating layers of high and low scattering in the parafoveal region reveal the stratified structure of the retina. The retinal nerve fiber layer (RNFL), consisting of horizontally propagating bundles of nerve axons, is visible as a bright backscattering layer adjacent to the vitreoretinal interface. The inner and outer plexiform layers (IPL, OPL) which, like the RNFL, correspond to axonic connections between neurons, also appear as regions of enhanced scattering. The IPL and OPL lie anterior to the the inner and outer result of the contour estimation and filtering process for the retinal image. It is evident that the spatial cut-off frequency of the low-pass filter determines the extent to which real contour variations are interpreted as unwanted motion, and vice versa. A spatial frequency cutoff of 0 cycles/image was selected for the nominally flat macular region displayed. Although the cross-correlation scan reregistration technique does not compensate for transverse ocular motion, these variations are most likely small compared to the transverse pixel size in all the tomographs presented in this article. High resolution OCT imaging of the foveal region, as shown in Fig. 4, is promising for the diagnosis and monitoring of macular disease. Macular edema, for example, manifested as increased retinal thickness, is the leading cause of blindness in diabetic retinopathy, and is commonly associated with vascular occlusions, postcataract extraction, and

uveitis. Current diagnostic techniques, including slit-lamp observation and fundus photography, are relatively insensitive to increases in retinal thickness [22]. Fluorescein leakage is also a poor indicator of fluid accumulation and subsequent loss in visual acuity [23]. OCT can provide images of retinal thickness with micron scale lateral and transverse resolution, potentially permitting increased diagnostic sensitivity and quantitative assessment of the degree and localization of retinal thickening. Age-related macular degeneration is the leading cause of new blindness in the elderly in industrialized countries [24]. Clinical diagnosis depends on the early detection of choroidal neovascular membranes, which often form before the onset of vision loss [25]. Although fluorescein angiography is highly sensitive to leakage through these permeable membranes, angiography may not always accurately localize the leakage source, thus preventing the accurate delivery of photocoagulation therapy [26]. The high transverse resolution of OCT may provide a useful adjunct to angiography by pinpointing the location of the anatomic defect and aiding in the delivery of efficient therapy. OCT imaging of the optic nerve head and peripapillary region shows significant promise for the early diagnosis and monitoring of glaucoma, the leading cause of blindness in the United States. A series of OCT tomographs taken at different radial planes through the optic nerve head and surrounding retina of a volunteer. The contour of each image has been filtered with a cutoff of 10 cycles/image to remove motion artifacts. In the 90 degree tomograph (taken perpendicular to the papillomacular axis), high backscattering is again visible from the RNFL and choroid. The RNFL expands towards the optic disk to occupy nearly the entire retinal thickness, and demonstrates the presence of the inferior and superior arcuate nerve fiber bundles. In comparison, the 0 degree tomograph (taken parallel to the papillomacular axis) exhibits a markedly thinner RNFL, consistent with the absence of well-defined fiber bundles. Additionally, the predominance of nerve fibers emerging from high density ganglion cells in the parafoveal region causes the temporally located nerve fiber layer to appear thicker than the opposite nasal RNFL, because an increased convergence of rods onto ganglion cells in the peripheral retina leads to a thinner nerve fiber layer. In all the radial tomographs, the surface contour and cupping of the optic disk are well visualized. The termination of the choroid at the lamina cribosa is also clearly delineated, providing a convenient landmark which one could use to make reproducible measurements of RNFL and retinal thickness at the nerve head margin. Quantitation of retinal and RNFL thickness in the peripapillary region is directly relevant to the early diagnosis and treatment of glaucoma. Tonometry and visual field testing, the current mainstays of diagnosis, are complicated by

the fact that intraocular pressure often does not reliably predict disease progression [27], and visual field defects occur only after irreversible damage to the nerve fiber layer [28]. Clinical observations of the eye using ophthalmoscopy are extremely subjective, and variability among observers is often great, even among experienced specialists [29]. Stereo fundus photography reduces this variability somewhat, but still lacks the quantitative measures necessary to track interval changes in the optic disc, which may be necessary for accurate diagnosis. Changes in RNFL and retinal thickness may be a sensitive indicator of glaucoma onset and may precede other indicators such as optic nerve head appearance or cupping [30]. Histological studies have shown that 50 percent of the retinal nerve fiber layer may atrophy before detection by standard clinical methods [31]. Thus, the unique capability of OCT for high-resolution tomography in the posterior eye and direct measurement of RNFL thickness could provide a significant advance in the early diagnosis, monitoring, and treatment of glaucomatous patients. and penetration depth of the photocoagulation bum. Several important features of the healing process are also documented. Regrowth and thickening of the epithelium are clearly seen above the lesion, while the endothelial cell layer remains intact and undamaged just below the photocoagulation. The capability of OCT for non-invasive assessment of histopathology in vivo may be important in the clinical evaluation and longitudinal study of diverse ocular disease in both humans and animal models. OCT can record both changes at the cellular level and differences in large scale morphology, showing significant promise as a potential adjunct to keratorefractive therapies such as LTK, where both realtime monitoring of the surgery in progress and post- operative follow-up may require evaluation of the gross corneal curvature as well as cell damage and photocoagulation penetration depth. As a research tool, OCT may be also beneficial in determining the LTK exposure parameters that provide stable alterations in corneal profile without endothelial cell damage.

We consider the following anycast field equations defined over an open bounded piece of network and /or feature space  $\Omega \subset R^d$ . They describe the dynamics of the mean anycast of each of  $p$  node populations.

$$\begin{cases} \left( \frac{d}{dt} + l_i \right) V_i(t, r) = \sum_{j=1}^p \int_{\Omega} J_{ij}(r, \bar{r}) S[(V_j(t - \tau_{ij}(r, \bar{r}), \bar{r}) - h_{ij})] d\bar{r} \\ \quad + I_i^{ext}(r, t), \quad t \geq 0, 1 \leq i \leq p, \\ V_i(t, r) = \phi_i(t, r) \quad t \in [-T, 0] \end{cases} \quad (1)$$

We give an interpretation of the various parameters and functions that appear in (1),  $\Omega$  is finite piece of nodes and/or feature space and is

represented as an open bounded set of  $R^d$ . The vector  $r$  and  $\bar{r}$  represent points in  $\Omega$ . The function  $S: R \rightarrow (0,1)$  is the normalized sigmoid function:

$$S(z) = \frac{1}{1 + e^{-z}} \quad (2)$$

It describes the relation between the input rate  $v_i$  of population  $i$  as a function of the packets potential, for example,  $V_i = v_i = S[\sigma_i(V_i - h_i)]$ . We note  $V$  the  $p$ -dimensional vector  $(V_1, \dots, V_p)$ . The  $p$  function  $\phi_i, i=1, \dots, p$ , represent the initial conditions, see below. We note  $\phi$  the  $p$ -dimensional vector  $(\phi_1, \dots, \phi_p)$ . The  $p$  function  $I_i^{ext}, i=1, \dots, p$ , represent external factors from other network areas. We note  $I^{ext}$  the  $p$ -dimensional vector  $(I_1^{ext}, \dots, I_p^{ext})$ . The  $p \times p$  matrix of functions  $J = \{J_{ij}\}_{i,j=1, \dots, p}$  represents the connectivity between populations  $i$  and  $j$ , see below. The  $p$  real values  $h_i, i=1, \dots, p$ , determine the threshold of activity for each population, that is, the value of the nodes potential corresponding to 50% of the maximal activity. The  $p$  real positive values  $\sigma_i, i=1, \dots, p$ , determine the slopes of the sigmoids at the origin. Finally the  $p$  real positive values  $l_i, i=1, \dots, p$ , determine the speed at which each anycast node potential decreases exponentially toward its real value. We also introduce the function  $S: R^p \rightarrow R^p$ , defined by  $S(x) = [S(\sigma_1(x_1 - h_1)), \dots, S(\sigma_p(x_p - h_p))]$ , and the diagonal  $p \times p$  matrix  $L_0 = \text{diag}(l_1, \dots, l_p)$ . Is the intrinsic dynamics of the population given by the linear response of data transfer.  $(\frac{d}{dt} + l_i)$  is replaced by  $(\frac{d}{dt} + l_i)^2$  to use

the alpha function response. We use  $(\frac{d}{dt} + l_i)$  for simplicity although our analysis applies to more general intrinsic dynamics. For the sake of generality, the propagation delays are not assumed to be identical for all populations, hence they are described by a matrix  $\tau(r, \bar{r})$  whose element  $\tau_{ij}(r, \bar{r})$  is the propagation delay between population  $j$  at  $\bar{r}$  and population  $i$  at  $r$ . The reason for this assumption is that it is still unclear from anycast if propagation delays are independent

of the populations. We assume for technical reasons that  $\tau$  is continuous, that is  $\tau \in C^0(\bar{\Omega}^2, R_+^{p \times p})$ . Moreover packet data indicate that  $\tau$  is not a symmetric function i.e.,  $\tau_{ij}(r, \bar{r}) \neq \tau_{ji}(\bar{r}, r)$ , thus no assumption is made about this symmetry unless otherwise stated. In order to compute the righthand side of (1), we need to know the node potential factor  $V$  on interval  $[-T, 0]$ . The value of  $T$  is obtained by considering the maximal delay:

$$\tau_m = \max_{i,j(r, \bar{r}) \in \Omega \times \Omega} \tau_{i,j}(r, \bar{r}) \quad (3)$$

Hence we choose  $T = \tau_m$

### A. Mathematical Framework

A convenient functional setting for the non-delayed packet field equations is to use the space  $F = L^2(\Omega, R^p)$  which is a Hilbert space endowed with the usual inner product:

$$\langle V, U \rangle_F = \sum_{i=1}^p \int_{\Omega} V_i(r) U_i(r) dr \quad (1)$$

To give a meaning to (1), we defined the history space  $C = C^0([-\tau_m, 0], F)$  with  $\|\phi\| = \sup_{t \in [-\tau_m, 0]} \|\phi(t)\|_F$ , which is the Banach phase space associated with equation (3). Using the notation  $V_t(\theta) = V(t + \theta), \theta \in [-\tau_m, 0]$ , we write (1) as

$$\begin{cases} V(t) = -L_0 V(t) + L_1 S(V_t) + I^{ext}(t), \\ V_0 = \phi \in C, \end{cases} \quad (2)$$

Where

$$\begin{cases} L_1: C \rightarrow F, \\ \phi \rightarrow \int_{\Omega} J(\cdot, \bar{r}) \phi(\bar{r}, -\tau(\cdot, \bar{r})) d\bar{r} \end{cases}$$

Is the linear continuous operator satisfying  $\|L_1\| \leq \|J\|_{L^2(\Omega^2, R^{p \times p})}$ . Notice that most of the papers on this subject assume  $\Omega$  infinite, hence requiring  $\tau_m = \infty$ .

**Proposition 1.0** If the following assumptions are satisfied.

1.  $J \in L^2(\Omega^2, R^{p \times p})$ ,
2. The external current  $I^{ext} \in C^0(R, F)$ ,
3.  $\tau \in C^0(\bar{\Omega}^2, R_+^{p \times p}), \sup_{\bar{\Omega}^2} \tau \leq \tau_m$ .

Then for any  $\phi \in C$ , there exists a unique solution  $V \in C^1([0, \infty), F) \cap C^0([-\tau_m, \infty), F)$  to (3)

Notice that this result gives existence on  $R_+$ , finite-time explosion is impossible for this delayed differential equation. Nevertheless, a particular solution could grow indefinitely, we now prove that this cannot happen.

### B. Boundedness of Solutions

A valid model of neural networks should only feature bounded packet node potentials.

**Theorem 1.0** All the trajectories are ultimately bounded by the same constant  $R$  if  $I \equiv \max_{t \in R^+} \|I^{ext}(t)\|_F < \infty$ .

*Proof* :Let us defined  $f : R \times C \rightarrow R^+$  as  $f(t, V_t) \stackrel{def}{=} \left\langle -L_0 V_t(0) + L_1 S(V_t) + I^{ext}(t), V(t) \right\rangle_F = \frac{1}{2} \frac{d\|V\|_F^2}{dt}$

We note  $l = \min_{i=1, \dots, p} l_i$

$$f(t, V_t) \leq -l \|V(t)\|_F^2 + (\sqrt{p|\Omega|} \|J\|_F + I) \|V(t)\|_F$$

Thus, if

$$\|V(t)\|_F \geq 2 \frac{\sqrt{p|\Omega|} \|J\|_F + I}{l} \stackrel{def}{=} R, f(t, V_t) \leq -\frac{lR^2}{2} \stackrel{def}{=} -\delta < 0$$

Let us show that the open route of  $F$  of center 0 and radius  $R, B_R$ , is stable under the dynamics of equation. We know that  $V(t)$  is defined for all  $t \geq 0$  and that  $f < 0$  on  $\partial B_R$ , the boundary of  $B_R$ . We consider three cases for the initial condition  $V_0$ . If  $\|V_0\|_C < R$  and set  $T = \sup\{t \mid \forall s \in [0, t], V(s) \in \overline{B_R}\}$ . Suppose that  $T \in R$ , then  $V(T)$  is defined and belongs to  $\overline{B_R}$ , the closure of  $B_R$ , because  $\overline{B_R}$  is closed, in effect to  $\partial B_R$ , we also have  $\frac{d}{dt} \|V\|_F^2 \Big|_{t=T} = f(T, V_T) \leq -\delta < 0$  because  $V(T) \in \partial B_R$ . Thus we deduce that for  $\varepsilon > 0$  and small enough,  $V(T + \varepsilon) \in \overline{B_R}$  which contradicts the definition of  $T$ . Thus  $T \notin R$  and  $\overline{B_R}$  is stable. Because  $f < 0$  on  $\partial B_R, V(0) \in \partial B_R$  implies that  $\forall t > 0, V(t) \in B_R$ . Finally we consider the case  $V(0) \in \overline{CB_R}$ . Suppose that  $\forall t > 0, V(t) \notin \overline{B_R}$ ,

then  $\forall t > 0, \frac{d}{dt} \|V\|_F^2 \leq -2\delta$ , thus  $\|V(t)\|_F$  is monotonically decreasing and reaches the value of  $R$  in finite time when  $V(t)$  reaches  $\partial B_R$ . This contradicts our assumption. Thus  $\exists T > 0 \mid V(T) \in B_R$ .

**Proposition 1.1** : Let  $s$  and  $t$  be measured simple functions on  $X$ . for  $E \in M$ , define

$$\phi(E) = \int_E s d\mu \quad (1)$$

Then  $\phi$  is a measure on  $M$ .

$$\int_X (s+t) d\mu = \int_X s d\mu + \int_X t d\mu \quad (2)$$

**Proof** : If  $s$  and if  $E_1, E_2, \dots$  are disjoint members of  $M$  whose union is  $E$ , the countable additivity of  $\mu$  shows that

$$\begin{aligned} \phi(E) &= \sum_{i=1}^n \alpha_i \mu(A_i \cap E) = \sum_{i=1}^n \alpha_i \sum_{r=1}^{\infty} \mu(A_i \cap E_r) \\ &= \sum_{r=1}^{\infty} \sum_{i=1}^n \alpha_i \mu(A_i \cap E_r) = \sum_{r=1}^{\infty} \phi(E_r) \end{aligned}$$

Also,  $\phi(\phi) = 0$ , so that  $\phi$  is not identically  $\infty$ .

Next, let  $s$  be as before, let  $\beta_1, \dots, \beta_m$  be the distinct values of  $t$ , and let  $B_j = \{x : t(x) = \beta_j\}$  If  $E_{ij} = A_i \cap B_j$ ,

$$\text{the } \int_{E_{ij}} (s+t) d\mu = (\alpha_i + \beta_j) \mu(E_{ij})$$

$$\text{and } \int_{E_{ij}} s d\mu + \int_{E_{ij}} t d\mu = \alpha_i \mu(E_{ij}) + \beta_j \mu(E_{ij})$$

Thus (2) holds with  $E_{ij}$  in place of  $X$ . Since  $X$  is the disjoint union of the sets  $E_{ij}$  ( $1 \leq i \leq n, 1 \leq j \leq m$ ), the first half of our proposition implies that (2) holds.

**Theorem 1.1:** If  $K$  is a compact set in the plane whose complement is connected, if  $f$  is a continuous complex function on  $K$  which is holomorphic in the interior of  $K$ , and if  $\varepsilon > 0$ , then there exists a polynomial  $P$  such that  $|f(z) - P(z)| < \varepsilon$  for all  $z \in K$ . If the interior of  $K$  is empty, then part of the hypothesis is vacuously satisfied, and the conclusion holds for every  $f \in C(K)$ . Note that  $K$  need to be connected.

*Proof:* By Tietze's theorem,  $f$  can be extended to a continuous function in the plane, with compact support. We fix one such extension and denote it

again by  $f$ . For any  $\delta > 0$ , let  $\omega(\delta)$  be the supremum of the numbers  $|f(z_2) - f(z_1)|$  Where  $z_1$  and  $z_2$  are subject to the condition  $|z_2 - z_1| \leq \delta$ . Since  $f$  is uniformly continuous, we have  $\lim_{\delta \rightarrow 0} \omega(\delta) = 0$  (1) From now on,

$\delta$  will be fixed. We shall prove that there is a polynomial  $P$  such that  $|f(z) - P(z)| < 10,000 \omega(\delta)$  ( $z \in K$ ) (2)

By (1), this proves the theorem. Our first objective is the construction of a function  $\Phi \in C_c^1(R^2)$ , such that for all  $z$

$$|f(z) - \Phi(z)| \leq \omega(\delta), \quad (3)$$

$$|(\partial\Phi)(z)| < \frac{2\omega(\delta)}{\delta}, \quad (4)$$

And

$$\Phi(z) = -\frac{1}{\pi} \iint_X \frac{(\partial\Phi)(\zeta)}{\zeta - z} d\zeta d\eta \quad (\zeta = \xi + i\eta), \quad (5)$$

Where  $X$  is the set of all points in the support of  $\Phi$  whose distance from the complement of  $K$  does not  $\delta$ . (Thus  $X$  contains no point which is "far within"  $K$ .) We construct  $\Phi$  as the convolution of  $f$  with a smoothing function  $A$ . Put  $a(r) = 0$  if  $r > \delta$ , put

$$a(r) = \frac{3}{\pi\delta^2} \left(1 - \frac{r^2}{\delta^2}\right)^2 \quad (0 \leq r \leq \delta), \quad (6)$$

And define

$$A(z) = a(|z|) \quad (7)$$

For all complex  $z$ . It is clear that  $A \in C_c^1(R^2)$ . We claim that

$$\iint_{R^2} A = 1, \quad (8)$$

$$\iint_{R^2} \partial A = 0, \quad (9)$$

$$\iint_{R^2} |\partial A| = \frac{24}{15\delta} < \frac{2}{\delta}, \quad (10)$$

The constants are so adjusted in (6) that (8) holds. (Compute the integral in polar coordinates), (9) holds simply because  $A$  has compact support. To compute (10), express  $\partial A$  in polar coordinates, and note that  $\frac{\partial A}{\partial \theta} = 0$ ,

$$\frac{\partial A}{\partial r} = -a',$$

Now define

$$\Phi(z) = \iint_{R^2} f(z - \zeta) A d\xi d\eta = \iint_{R^2} A(z - \zeta) f(\zeta) d\xi d\eta \quad (11)$$

Since  $f$  and  $A$  have compact support, so does  $\Phi$ .

Since

$$\Phi(z) - f(z) = \iint_{R^2} [f(z - \zeta) - f(z)] A(\xi) d\xi d\eta \quad (12)$$

And  $A(\zeta) = 0$  if  $|\zeta| > \delta$ , (3) follows from (8).

The difference quotients of  $A$  converge boundedly to the corresponding partial derivatives, since  $A \in C_c^1(R^2)$ . Hence the last expression in (11) may be differentiated under the integral sign, and we obtain

$$\begin{aligned} (\partial\Phi)(z) &= \iint_{R^2} (\partial A)(z - \zeta) f(\zeta) d\xi d\eta \\ &= \iint_{R^2} f(z - \zeta) (\partial A)(\zeta) d\xi d\eta \\ &= \iint_{R^2} [f(z - \zeta) - f(z)] (\partial A)(\zeta) d\xi d\eta \end{aligned} \quad (13)$$

The last equality depends on (9). Now (10) and (13) give (4). If we write (13) with  $\Phi_x$  and  $\Phi_y$  in place of  $\partial\Phi$ , we see that  $\Phi$  has continuous partial derivatives, if we can show that  $\partial\Phi = 0$  in  $G$ , where  $G$  is the set of all  $z \in K$  whose distance from the complement of  $K$  exceeds  $\delta$ . We shall do this by showing that

$$\Phi(z) = f(z) \quad (z \in G); \quad (14)$$

Note that  $\partial f = 0$  in  $G$ , since  $f$  is holomorphic there. Now if  $z \in G$ , then  $z - \zeta$  is in the interior of  $K$  for all  $\zeta$  with  $|\zeta| < \delta$ . The mean value property for harmonic functions therefore gives, by the first equation in (11),

$$\begin{aligned} \Phi(z) &= \int_0^\delta a(r) r dr \int_0^{2\pi} f(z - re^{i\theta}) d\theta \\ &= 2\pi f(z) \int_0^\delta a(r) r dr = f(z) \iint_{R^2} A = f(z) \end{aligned} \quad (15)$$

For all  $z \in G$ , we have now proved (3), (4), and (5) The definition of  $X$  shows that  $X$  is compact and that  $X$  can be covered by finitely many open discs  $D_1, \dots, D_n$ , of radius  $2\delta$ , whose centers are not in  $K$ . Since  $S^2 - K$  is connected, the center of each  $D_j$  can be joined to  $\infty$  by a polygonal path in  $S^2 - K$ . It follows that each  $D_j$  contains a compact connected set  $E_j$ , of diameter at least  $2\delta$ , so that  $S^2 - E_j$  is connected

and so that  $K \cap E_j = \emptyset$ . with  $r = 2\delta$ . There are functions  $g_j \in H(S^2 - E_j)$  and constants  $b_j$  so that the inequalities.

$$|Q_j(\zeta, z)| < \frac{50}{\delta}, \quad (16)$$

$$\left| Q_j(\zeta, z) - \frac{1}{z - \zeta} \right| < \frac{4,000\delta^2}{|z - \zeta|^2} \quad (17)$$

Hold for  $z \notin E_j$  and  $\zeta \in D_j$ , if

$$Q_j(\zeta, z) = g_j(z) + (\zeta - b_j)g_j^2(z) \quad (18)$$

Let  $\Omega$  be the complement of  $E_1 \cup \dots \cup E_n$ . Then

$\Omega$  is an open set which contains  $K$ . Put

$$X_1 = X \cap D_1 \quad \text{and}$$

$$X_j = (X \cap D_j) - (X_1 \cup \dots \cup X_{j-1}), \quad \text{for}$$

$$2 \leq j \leq n,$$

Define

$$R(\zeta, z) = Q_j(\zeta, z) \quad (\zeta \in X_j, z \in \Omega) \quad (19)$$

And

$$F(z) = \frac{1}{\pi} \iint_X (\partial\Phi)(\zeta) R(\zeta, z) d\zeta d\eta \quad (20)$$

$$(z \in \Omega)$$

Since,

$$F(z) = \sum_{j=1}^n \frac{1}{\pi} \iint_{X_j} (\partial\Phi)(\zeta) Q_j(\zeta, z) d\zeta d\eta, \quad (21)$$

(18) shows that  $F$  is a finite linear combination of the functions  $g_j$  and  $g_j^2$ . Hence  $F \in H(\Omega)$ . By (20), (4), and (5) we have

$$|F(z) - \Phi(z)| < \frac{2\omega(\delta)}{\pi\delta} \iint_X |R(\zeta, z)|$$

$$- \frac{1}{z - \zeta} |d\zeta d\eta \quad (z \in \Omega) \quad (22)$$

Observe that the inequalities (16) and (17) are valid with  $R$  in place of  $Q_j$  if  $\zeta \in X$  and  $z \in \Omega$ . Now fix  $z \in \Omega$ , put  $\zeta = z + \rho e^{i\theta}$ , and estimate the integrand in (22) by (16) if  $\rho < 4\delta$ , by (17) if  $4\delta \leq \rho$ . The integral in (22) is then seen to be less than the sum of

$$2\pi \int_0^{4\delta} \left( \frac{50}{\delta} + \frac{1}{\rho} \right) \rho d\rho = 808\pi\delta \quad (23)$$

And

$$2\pi \int_{4\delta}^{\infty} \frac{4,000\delta^2}{\rho^2} \rho d\rho = 2,000\pi\delta. \quad (24)$$

Hence (22) yields

$$|F(z) - \Phi(z)| < 6,000\omega(\delta) \quad (z \in \Omega) \quad (25)$$

Since  $F \in H(\Omega)$ ,  $K \subset \Omega$ , and  $S^2 - K$  is connected, Runge's theorem shows that  $F$  can be uniformly approximated on  $K$  by polynomials. Hence (3) and (25) show that (2) can be satisfied. This completes the proof.

**Lemma 1.0 :** Suppose  $f \in C_c'(R^2)$ , the space of all continuously differentiable functions in the plane, with compact support. Put

$$\partial = \frac{1}{2} \left( \frac{\partial}{\partial x} + i \frac{\partial}{\partial y} \right) \quad (1)$$

Then the following "Cauchy formula" holds:

$$f(z) = -\frac{1}{\pi} \iint_{R^2} \frac{(\partial f)(\zeta)}{\zeta - z} d\xi d\eta$$

$$(\zeta = \xi + i\eta) \quad (2)$$

**Proof:** This may be deduced from Green's theorem. However, here is a simple direct proof:

Put  $\varphi(r, \theta) = f(z + re^{i\theta})$ ,  $r > 0$ ,  $\theta$  real

If  $\zeta = z + re^{i\theta}$ , the chain rule gives

$$(\partial f)(\zeta) = \frac{1}{2} e^{i\theta} \left[ \frac{\partial}{\partial r} + \frac{i}{r} \frac{\partial}{\partial \theta} \right] \varphi(r, \theta) \quad (3)$$

The right side of (2) is therefore equal to the limit, as  $\varepsilon \rightarrow 0$ , of

$$-\frac{1}{2} \int_{\varepsilon}^{\infty} \int_0^{2\pi} \left( \frac{\partial \varphi}{\partial r} + \frac{i}{r} \frac{\partial \varphi}{\partial \theta} \right) d\theta dr \quad (4)$$

For each  $r > 0$ ,  $\varphi$  is periodic in  $\theta$ , with period  $2\pi$ . The integral of  $\partial\varphi / \partial\theta$  is therefore 0, and (4) becomes

$$-\frac{1}{2\pi} \int_0^{2\pi} d\theta \int_{\varepsilon}^{\infty} \frac{\partial \varphi}{\partial r} dr = \frac{1}{2\pi} \int_0^{2\pi} \varphi(\varepsilon, \theta) d\theta \quad (5)$$

As  $\varepsilon \rightarrow 0$ ,  $\varphi(\varepsilon, \theta) \rightarrow f(z)$  uniformly. This gives (2)

If  $X^\alpha \in a$  and  $X^\beta \in k[X_1, \dots, X_n]$ , then

$X^\alpha X^\beta = X^{\alpha+\beta} \in a$ , and so  $A$  satisfies the condition (\*). Conversely,

$$\left( \sum_{\alpha \in A} c_\alpha X^\alpha \right) \left( \sum_{\beta \in \square^n} d_\beta X^\beta \right) = \sum_{\alpha, \beta} c_\alpha d_\beta X^{\alpha+\beta} \quad (\text{finite sums}),$$

and so if  $A$  satisfies (\*), then the subspace generated by the monomials  $X^\alpha, \alpha \in a$ , is an ideal. The proposition gives a classification of the monomial ideals in  $k[X_1, \dots, X_n]$ : they are in one to one correspondence with the subsets  $A$  of  $\square^n$  satisfying (\*). For example, the monomial ideals in

$k[X]$  are exactly the ideals  $(X^n)$ ,  $n \geq 1$ , and the zero ideal (corresponding to the empty set  $A$ ). We write  $\langle X^\alpha \mid \alpha \in A \rangle$  for the ideal corresponding to  $A$  (subspace generated by the  $X^\alpha, \alpha \in A$ ).

**LEMMA 1.1.** Let  $S$  be a subset of  $\square^n$ . The ideal  $a$  generated by  $X^\alpha, \alpha \in S$  is the monomial ideal corresponding to

$$A \stackrel{\text{df}}{=} \{ \beta \in \square^n \mid \beta - \alpha \in \square^n, \text{ some } \alpha \in S \}$$

Thus, a monomial is in  $a$  if and only if it is divisible by one of the  $X^\alpha, \alpha \in S$

**PROOF.** Clearly  $A$  satisfies  $(*)$ , and  $a \subset \langle X^\beta \mid \beta \in A \rangle$ . Conversely, if  $\beta \in A$ , then  $\beta - \alpha \in \square^n$  for some  $\alpha \in S$ , and  $X^\beta = X^\alpha X^{\beta-\alpha} \in a$ . The last statement follows from the fact that  $X^\alpha \mid X^\beta \Leftrightarrow \beta - \alpha \in \square^n$ . Let

$A \subset \square^n$  satisfy  $(*)$ . From the geometry of  $A$ , it is clear that there is a finite set of elements  $S = \{ \alpha_1, \dots, \alpha_s \}$  of  $A$  such that  $A = \{ \beta \in \square^n \mid \beta - \alpha_i \in \square^2, \text{ some } \alpha_i \in S \}$

(The  $\alpha_i$ 's are the corners of  $A$ ) Moreover,  $a \stackrel{\text{df}}{=} \langle X^\alpha \mid \alpha \in A \rangle$  is generated by the monomials  $X^{\alpha_i}, \alpha_i \in S$ .

**DEFINITION 1.0.** For a nonzero ideal  $a$  in  $k[X_1, \dots, X_n]$ , we let  $(LT(a))$  be the ideal generated by  $\{ LT(f) \mid f \in a \}$

**LEMMA 1.2** Let  $a$  be a nonzero ideal in  $k[X_1, \dots, X_n]$ ; then  $(LT(a))$  is a monomial ideal, and it equals  $(LT(g_1), \dots, LT(g_n))$  for some  $g_1, \dots, g_n \in a$ .

**PROOF.** Since  $(LT(a))$  can also be described as the ideal generated by the leading monomials (rather than the leading terms) of elements of  $a$ .

**THEOREM 1.2.** Every ideal  $a$  in  $k[X_1, \dots, X_n]$  is finitely generated; more precisely,  $a = (g_1, \dots, g_s)$  where  $g_1, \dots, g_s$  are any

elements of  $a$  whose leading terms generate  $LT(a)$

**PROOF.** Let  $f \in a$ . On applying the division algorithm, we find  $f = a_1 g_1 + \dots + a_s g_s + r$ ,  $a_i, r \in k[X_1, \dots, X_n]$ , where either  $r = 0$  or no monomial occurring in it is divisible by any  $LT(g_i)$ . But  $r = f - \sum a_i g_i \in a$ , and therefore  $LT(r) \in LT(a) = (LT(g_1), \dots, LT(g_s))$ , implies that every monomial occurring in  $r$  is divisible by one in  $LT(g_i)$ . Thus  $r = 0$ , and  $g \in (g_1, \dots, g_s)$ .

**DEFINITION 1.1.** A finite subset  $S = \{ g_1, \dots, g_s \}$  of an ideal  $a$  is a standard (*Gröbner*) bases for  $a$  if  $(LT(g_1), \dots, LT(g_s)) = LT(a)$ . In other words,  $S$  is a standard basis if the leading term of every element of  $a$  is divisible by at least one of the leading terms of the  $g_i$ .

**THEOREM 1.3** The ring  $k[X_1, \dots, X_n]$  is Noetherian i.e., every ideal is finitely generated.

**PROOF.** For  $n = 1$ ,  $k[X]$  is a principal ideal domain, which means that every ideal is generated by single element. We shall prove the theorem by induction on  $n$ . Note that the obvious map  $k[X_1, \dots, X_{n-1}][X_n] \rightarrow k[X_1, \dots, X_n]$  is an isomorphism – this simply says that every polynomial  $f$  in  $n$  variables  $X_1, \dots, X_n$  can be expressed uniquely as a polynomial in  $X_n$  with coefficients in  $k[X_1, \dots, X_{n-1}]$ :

$$f(X_1, \dots, X_n) = a_0(X_1, \dots, X_{n-1})X_n^r + \dots + a_r(X_1, \dots, X_{n-1})$$

Thus the next lemma will complete the proof

**LEMMA 1.3.** If  $A$  is Noetherian, then so also is  $A[X]$

**PROOF.** For a polynomial

$$f(X) = a_0 X^r + a_1 X^{r-1} + \dots + a_r, \quad a_i \in A, \quad a_0 \neq 0,$$

$r$  is called the degree of  $f$ , and  $a_0$  is its leading coefficient. We call  $0$  the leading coefficient of the polynomial  $0$ . Let  $a$  be an ideal in  $A[X]$ . The

leading coefficients of the polynomials in  $a$  form an ideal  $a'$  in  $A$ , and since  $A$  is Noetherian,  $a'$  will be finitely generated. Let  $g_1, \dots, g_m$  be elements of  $a$  whose leading coefficients generate  $a'$ , and let  $r$  be the maximum degree of  $g_i$ . Now let  $f \in a$ , and suppose  $f$  has degree  $s > r$ , say,  $f = aX^s + \dots$ . Then  $a \in a'$ , and so we can write  $a = \sum b_i a_i$ ,  $b_i \in A$ ,

$a_i = \text{leading coefficient of } g_i$

Now

$f - \sum b_i g_i X^{s-r_i}$ ,  $r_i = \text{deg}(g_i)$ , has degree  $< \text{deg}(f)$ . By continuing in this way, we find that  $f \equiv f_t \pmod{(g_1, \dots, g_m)}$  With  $f_t$  a polynomial of degree  $t < r$ . For each  $d < r$ , let  $a_d$  be the subset of  $A$  consisting of 0 and the leading coefficients of all polynomials in  $a$  of degree  $d$ ; it is again an ideal in  $A$ . Let  $g_{d,1}, \dots, g_{d,m_d}$  be polynomials of degree  $d$  whose leading coefficients generate  $a_d$ . Then the same argument as above shows that any polynomial  $f_d$  in  $a$  of degree  $d$  can be written  $f_d \equiv f_{d-1} \pmod{(g_{d,1}, \dots, g_{d,m_d})}$  With  $f_{d-1}$  of degree  $\leq d-1$ . On applying this remark repeatedly we find that  $f_t \in (g_{r-1,1}, \dots, g_{r-1,m_{r-1}}, \dots, g_{0,1}, \dots, g_{0,m_0})$  Hence

$$f_t \in (g_1, \dots, g_m, g_{r-1,1}, \dots, g_{r-1,m_{r-1}}, \dots, g_{0,1}, \dots, g_{0,m_0})$$

and so the polynomials  $g_1, \dots, g_{0,m_0}$  generate  $a$

One of the great successes of category theory in computer science has been the development of a "unified theory" of the constructions underlying denotational semantics. In the untyped  $\lambda$ -calculus, any term may appear in the function position of an application. This means that a model  $D$  of the  $\lambda$ -calculus must have the property that given a term  $t$  whose interpretation is  $d \in D$ , Also, the interpretation of a functional abstraction like  $\lambda x. x$  is most conveniently defined as a function from  $D$  to  $D$ , which must then be regarded as an element of  $D$ . Let  $\psi: [D \rightarrow D] \rightarrow D$  be the function that picks out elements of  $D$  to represent elements of  $[D \rightarrow D]$  and  $\phi: D \rightarrow [D \rightarrow D]$  be the function that maps

elements of  $D$  to functions of  $D$ . Since  $\psi(f)$  is intended to represent the function  $f$  as an element of  $D$ , it makes sense to require that  $\phi(\psi(f)) = f$ , that is,  $\psi \circ \psi = id_{[D \rightarrow D]}$  Furthermore, we often want to view every element of  $D$  as representing some function from  $D$  to  $D$  and require that elements representing the same function be equal – that is  $\psi(\phi(d)) = d$

or

$$\psi \circ \phi = id_D$$

The latter condition is called extensionality.

These conditions together imply that  $\phi$  and  $\psi$  are inverses--- that is,  $D$  is isomorphic to the space of functions from  $D$  to  $D$  that can be the interpretations of functional abstractions:  $D \cong [D \rightarrow D]$ . Let us suppose we are working with the untyped  $\lambda$ -calculus, we need a solution of the equation  $D \cong A + [D \rightarrow D]$ , where  $A$  is some predetermined domain containing interpretations for elements of  $C$ . Each element of  $D$  corresponds to either an element of  $A$  or an element of  $[D \rightarrow D]$ , with a tag. This equation can be solved by finding least fixed points of the function  $F(X) = A + [X \rightarrow X]$  from domains to domains --- that is, finding domains  $X$  such that  $X \cong A + [X \rightarrow X]$ , and such that for any domain  $Y$  also satisfying this equation, there is an embedding of  $X$  to  $Y$  --- a pair of maps

$$X \begin{matrix} \xrightarrow{f} \\ \square \\ \xrightarrow{f^R} \end{matrix} Y$$

Such that

$$f^R \circ f = id_X$$

$$f \circ f^R \subseteq id_Y$$

Where  $f \subseteq g$  means that

$f$  approximates  $g$  in some ordering representing their information content. The key shift of perspective from the domain-theoretic to the more general category-theoretic approach lies in considering  $F$  not as a function on domains, but as a functor on a category of domains. Instead of a least fixed point of the function,  $F$ .

**Definition 1.3:** Let  $K$  be a category and  $F: K \rightarrow K$  as a functor. A fixed point of  $F$  is a pair  $(A, a)$ , where  $A$  is a **K-object** and  $a: F(A) \rightarrow A$  is an isomorphism. A prefixed point of  $F$  is a pair  $(A, a)$ , where  $A$  is a **K-object** and  $a$  is any arrow from  $F(A)$  to  $A$

**Definition 1.4 :** An  $\omega$ -chain in a category  $\mathbf{K}$  is a diagram of the following form:

$$\Delta = D_0 \xrightarrow{f_0} D_1 \xrightarrow{f_1} D_2 \xrightarrow{f_2} \dots$$

Recall that a cocone  $\mu$  of an  $\omega$ -chain  $\Delta$  is a  $\mathbf{K}$ -object  $X$  and a collection of  $\mathbf{K}$ -arrows  $\{\mu_i : D_i \rightarrow X \mid i \geq 0\}$  such that  $\mu_i = \mu_{i+1} \circ f_i$  for all  $i \geq 0$ . We sometimes write  $\mu : \Delta \rightarrow X$  as a reminder of the arrangement of  $\mu$ 's components. Similarly, a colimit  $\mu : \Delta \rightarrow X$  is a cocone with the property that if  $\nu : \Delta \rightarrow X'$  is also a cocone then there exists a unique mediating arrow  $k : X \rightarrow X'$  such that for all  $i \geq 0$ ,  $\nu_i = k \circ \mu_i$ . Colimits of  $\omega$ -chains are sometimes referred to as  $\omega$ -colimits. Dually, an  $\omega^{op}$ -chain in  $\mathbf{K}$  is a diagram of the following form:

$$\Delta = D_0 \xleftarrow{f_0} D_1 \xleftarrow{f_1} D_2 \xleftarrow{f_2} \dots$$

A cone  $\mu : X \rightarrow \Delta$  of an  $\omega^{op}$ -chain  $\Delta$  is a  $\mathbf{K}$ -object  $X$  and a collection of  $\mathbf{K}$ -arrows  $\{\mu_i : D_i \rightarrow X \mid i \geq 0\}$  such that for all  $i \geq 0$ ,  $\mu_i = f_i \circ \mu_{i+1}$ . An  $\omega^{op}$ -limit of an  $\omega^{op}$ -chain  $\Delta$  is a cone  $\mu : X \rightarrow \Delta$  with the property that if  $\nu : X' \rightarrow \Delta$  is also a cone, then there exists a unique mediating arrow  $k : X' \rightarrow X$  such that for all  $i \geq 0$ ,  $\mu_i \circ k = \nu_i$ . We write  $\perp_k$  (or just  $\perp$ ) for the distinguished initial object of  $\mathbf{K}$ , when it has one, and  $\perp \rightarrow A$  for the unique arrow from  $\perp$  to each  $\mathbf{K}$ -object  $A$ . It is also convenient to write  $\Delta^- = D_1 \xrightarrow{f_1} D_2 \xrightarrow{f_2} \dots$  to denote all of  $\Delta$  except  $D_0$  and  $f_0$ . By analogy,  $\mu^-$  is  $\{\mu_i \mid i \geq 1\}$ .

For the images of  $\Delta$  and  $\mu$  under  $F$  we write  $F(\Delta) = F(D_0) \xrightarrow{F(f_0)} F(D_1) \xrightarrow{F(f_1)} F(D_2) \xrightarrow{F(f_2)} \dots$  and  $F(\mu) = \{F(\mu_i) \mid i \geq 0\}$

We write  $F^i$  for the  $i$ -fold iterated composition of  $F$  that is,  $F^0(f) = f, F^1(f) = F(f), F^2(f) = F(F(f))$ , etc.

With these definitions we can state that every monotonic function on a complete lattice has a least fixed point:

**Lemma 1.4.** Let  $\mathbf{K}$  be a category with initial object  $\perp$  and let  $F : \mathbf{K} \rightarrow \mathbf{K}$  be a functor. Define the  $\omega$ -chain  $\Delta$  by

$$\Delta = \perp \xrightarrow{\perp \rightarrow F(\perp)} F(\perp) \xrightarrow{F(\perp \rightarrow F(\perp))} F^2(\perp) \xrightarrow{F^2(\perp \rightarrow F(\perp))} \dots$$

If both  $\mu : \Delta \rightarrow D$  and  $F(\mu) : F(\Delta) \rightarrow F(D)$  are colimits, then  $(D, d)$  is an initial  $F$ -algebra, where  $d : F(D) \rightarrow D$  is the mediating arrow from  $F(\mu)$  to the cocone  $\mu^-$

**Theorem 1.4** Let a DAG  $G$  given in which each node is a random variable, and let a discrete conditional probability distribution of each node given values of its parents in  $G$  be specified. Then the product of these conditional distributions yields a joint probability distribution  $P$  of the variables, and  $(G, P)$  satisfies the Markov condition.

**Proof.** Order the nodes according to an ancestral ordering. Let  $X_1, X_2, \dots, X_n$  be the resultant ordering. Next define.

$$P(x_1, x_2, \dots, x_n) = P(x_n \mid pa_n) P(x_{n-1} \mid pa_{n-1}) \dots P(x_2 \mid pa_2) P(x_1 \mid pa_1),$$

Where  $PA_i$  is the set of parents of  $X_i$  of in  $G$  and  $P(x_i \mid pa_i)$  is the specified conditional probability distribution. First we show this does indeed yield a joint probability distribution. Clearly,  $0 \leq P(x_1, x_2, \dots, x_n) \leq 1$  for all values of the variables. Therefore, to show we have a joint distribution, as the variables range through all their possible values, is equal to one. To that end, specified conditional distributions are the conditional distributions they notationally represent in the joint distribution. Finally, we show the Markov condition is satisfied. To do this, we need show for  $1 \leq k \leq n$  that whenever

$$P(pa_k) \neq 0, \text{ if } P(nd_k \mid pa_k) \neq 0$$

$$\text{and } P(x_k \mid pa_k) \neq 0$$

$$\text{then } P(x_k \mid nd_k, pa_k) = P(x_k \mid pa_k),$$

Where  $ND_k$  is the set of nondescendants of  $X_k$  of in  $G$ . Since  $PA_k \subseteq ND_k$ , we need only show  $P(x_k \mid nd_k) = P(x_k \mid pa_k)$ . First for a given  $k$ , order the nodes so that all and only nondescendants of  $X_k$  precede  $X_k$  in the ordering. Note that this ordering depends on  $k$ , whereas the ordering in the first part of the proof does not. Clearly then

$$ND_k = \{X_1, X_2, \dots, X_{k-1}\}$$

Let

$$D_k = \{X_{k+1}, X_{k+2}, \dots, X_n\}$$

follows  $\sum_{d_k}$

We define the  $m^{\text{th}}$  cyclotomic field to be the field  $Q[x]/(\Phi_m(x))$  Where  $\Phi_m(x)$  is the  $m^{\text{th}}$  cyclotomic polynomial.  $Q[x]/(\Phi_m(x))$  has degree  $\varphi(m)$  over  $Q$  since  $\Phi_m(x)$  has degree  $\varphi(m)$ . The roots of  $\Phi_m(x)$  are just the primitive  $m^{\text{th}}$  roots of unity, so the complex embeddings of  $Q[x]/(\Phi_m(x))$  are simply the  $\varphi(m)$  maps

$$\sigma_k : Q[x]/(\Phi_m(x)) \mapsto C,$$

$$1 \leq k < m, (k, m) = 1, \text{ where}$$

$$\sigma_k(x) = \xi_m^k,$$

$\xi_m$  being our fixed choice of primitive  $m^{\text{th}}$  root of unity. Note that  $\xi_m^k \in Q(\xi_m)$  for every  $k$ ; it follows that  $Q(\xi_m) = Q(\xi_m^k)$  for all  $k$  relatively prime to  $m$ . In particular, the images of the  $\sigma_i$  coincide, so  $Q[x]/(\Phi_m(x))$  is Galois over  $Q$ . This means that we can write  $Q(\xi_m)$  for  $Q[x]/(\Phi_m(x))$  without much fear of ambiguity; we will do so from now on, the identification being  $\xi_m \mapsto x$ . One advantage of this is that one can easily talk about cyclotomic fields being extensions of one another, or intersections or compositums; all of these things take place considering them as subfield of  $C$ . We now investigate some basic properties of cyclotomic fields. The first issue is whether or not they are all distinct; to determine this, we need to know which roots of unity lie in  $Q(\xi_m)$ . Note, for example, that if  $m$  is odd, then  $-\xi_m$  is a  $2m^{\text{th}}$  root of unity. We will show that this is the only way in which one can obtain any non- $m^{\text{th}}$  roots of unity.

**LEMMA 1.5** If  $m$  divides  $n$ , then  $Q(\xi_m)$  is contained in  $Q(\xi_n)$

**PROOF.** Since  $\xi_m^{n/m} = \xi_m$ , we have  $\xi_m \in Q(\xi_n)$ , so the result is clear

**LEMMA 1.6** If  $m$  and  $n$  are relatively prime, then

$$Q(\xi_m, \xi_n) = Q(\xi_{mn})$$

and

$$Q(\xi_m) \cap Q(\xi_n) = Q$$

(Recall the  $Q(\xi_m, \xi_n)$  is the compositum of  $Q(\xi_m)$  and  $Q(\xi_n)$ )

**PROOF.** One checks easily that  $\xi_m \xi_n$  is a primitive  $mn^{\text{th}}$  root of unity, so that

$$Q(\xi_{mn}) \subseteq Q(\xi_m, \xi_n)$$

$$[Q(\xi_m, \xi_n) : Q] \leq [Q(\xi_m) : Q][Q(\xi_n) : Q] \\ = \varphi(m)\varphi(n) = \varphi(mn);$$

Since  $[Q(\xi_{mn}) : Q] = \varphi(mn)$ ; this implies that

$Q(\xi_m, \xi_n) = Q(\xi_{mn})$  We know that  $Q(\xi_m, \xi_n)$  has degree  $\varphi(mn)$  over  $Q$ , so we must have

$$[Q(\xi_m, \xi_n) : Q(\xi_m)] = \varphi(n)$$

and

$$[Q(\xi_m, \xi_n) : Q(\xi_n)] = \varphi(m)$$

$$[Q(\xi_m) : Q(\xi_m) \cap Q(\xi_n)] \geq \varphi(m)$$

And thus that  $Q(\xi_m) \cap Q(\xi_n) = Q$

**PROPOSITION 1.2** For any  $m$  and  $n$

$$Q(\xi_m, \xi_n) = Q(\xi_{[m,n]})$$

And

$$Q(\xi_m) \cap Q(\xi_n) = Q(\xi_{(m,n)});$$

here  $[m, n]$  and  $(m, n)$  denote the least common multiple and the greatest common divisor of  $m$  and  $n$ , respectively.

**PROOF.**

Write

$m = p_1^{e_1} \dots p_k^{e_k}$  and  $p_1^{f_1} \dots p_k^{f_k}$  where the  $p_i$  are distinct primes. (We allow  $e_i$  or  $f_i$  to be zero)

$$Q(\xi_m) = Q(\xi_{p_1^{e_1}})Q(\xi_{p_2^{e_2}}) \dots Q(\xi_{p_k^{e_k}})$$

and

$$Q(\xi_n) = Q(\xi_{p_1^{f_1}})Q(\xi_{p_2^{f_2}}) \dots Q(\xi_{p_k^{f_k}})$$

Thus

$$Q(\xi_m, \xi_n) = Q(\xi_{p_1^{e_1}}) \dots Q(\xi_{p_2^{e_2}}) Q(\xi_{p_1^{f_1}}) \dots Q(\xi_{p_k^{f_k}}) \\ = Q(\xi_{p_1^{e_1}})Q(\xi_{p_1^{f_1}}) \dots Q(\xi_{p_k^{e_k}})Q(\xi_{p_k^{f_k}}) \\ = Q(\xi_{p_1^{\max(e_1, f_1)}}) \dots Q(\xi_{p_k^{\max(e_k, f_k)}}) \\ = Q(\xi_{p_1^{\max(e_1, f_1)} \dots p_k^{\max(e_k, f_k)}}) \\ = Q(\xi_{[m,n]});$$

An entirely similar computation shows that  $Q(\xi_m) \cap Q(\xi_n) = Q(\xi_{(m,n)})$

Mutual information measures the information transferred when  $x_i$  is sent and  $y_i$  is received, and is defined as

$$I(x_i, y_i) = \log_2 \frac{P(x_i/y_i)}{P(x_i)} \text{ bits} \quad (1)$$

In a noise-free channel, **each**  $y_i$  is uniquely connected to the corresponding  $x_i$ , and so they constitute an input-output pair  $(x_i, y_i)$  for which

$$P(x_i/y_i) = 1 \text{ and } I(x_i, y_i) = \log_2 \frac{1}{P(x_i)} \text{ bits;}$$

that is, the transferred information is equal to the self-information that corresponds to the input  $x_i$ . In a

very noisy channel, the output  $y_i$  and input  $x_i$  would be completely uncorrelated, and so

$$P(x_i/y_i) = P(x_i) \text{ and also } I(x_i, y_i) = 0; \text{ that is,}$$

there is no transference of information. In general, a given channel will operate between these two extremes. The mutual information is defined between the input and the output of a given channel. An average of the calculation of the mutual information for all input-output pairs of a given channel is the average mutual information:

$$I(X, Y) = \sum_{i,j} P(x_i, y_j) I(x_i, y_j) = \sum_{i,j} P(x_i, y_j) \log_2 \left[ \frac{P(x_i/y_j)}{P(x_i)} \right]$$

bits per symbol. This calculation is done over the input and output alphabets. The average mutual information. The following expressions are useful for modifying the mutual information expression:

$$P(x_i, y_j) = P(x_i/y_j)P(y_j) = P(y_j/x_i)P(x_i)$$

$$P(y_j) = \sum_i P(y_j/x_i)P(x_i)$$

$$P(x_i) = \sum_j P(x_i/y_j)P(y_j)$$

Then

$$I(X, Y) = \sum_{i,j} P(x_i, y_j) \log_2 \left[ \frac{1}{P(x_i)} \right]$$

$$- \sum_{i,j} P(x_i, y_j) \log_2 \left[ \frac{1}{P(x_i/y_j)} \right]$$

$$= \sum_{i,j} P(x_i, y_j) \log_2 \left[ \frac{1}{P(x_i)} \right] = \sum_i \left[ P(x_i/y_i)P(y_j) \right] \log_2 \frac{1}{P(x_i)}$$

$$\sum_i P(x_i) \log_2 \frac{1}{P(x_i)} = H(X)$$

$$I(X, Y) = H(X) - H(X/Y)$$

$$\text{Where } H(X/Y) = \sum_{i,j} P(x_i, y_j) \log_2 \frac{1}{P(x_i/y_j)}$$

is usually called the equivocation. In a sense, the equivocation can be seen as the information lost in the noisy channel, and is a function of the backward conditional probability. The observation of an output symbol  $y_j$  provides  $H(X) - H(X/Y)$  bits of information. This difference is the mutual information of the channel. *Mutual Information: Properties* Since

$$P(x_i/y_j)P(y_j) = P(y_j/x_i)P(x_i)$$

The mutual information fits the condition

$$I(X, Y) = I(Y, X)$$

And by interchanging input and output it is also true that

$$I(X, Y) = H(Y) - H(Y/X)$$

Where

$$H(Y) = \sum_j P(y_j) \log_2 \frac{1}{P(y_j)}$$

This last entropy is usually called the noise entropy. Thus, the information transferred through the channel is the difference between the output entropy and the noise entropy. Alternatively, it can be said that the channel mutual information is the difference between the number of bits needed for determining a given input symbol before knowing the corresponding output symbol, and the number of bits needed for determining a given input symbol

after knowing the corresponding output symbol

$$I(X, Y) = H(X) - H(X/Y)$$

As the channel mutual information expression is a difference between two quantities, it seems that this parameter can adopt negative values. However, and in spite of the fact that for some  $y_j$ ,  $H(X/y_j)$  can be larger than  $H(X)$ , this is not possible for the average value calculated over all the outputs:

$$\sum_{i,j} P(x_i, y_j) \log_2 \frac{P(x_i/y_j)}{P(x_i)} = \sum_{i,j} P(x_i, y_j) \log_2 \frac{P(x_i, y_j)}{P(x_i)P(y_j)}$$

Then

$$-I(X, Y) = \sum_{i,j} P(x_i, y_j) \frac{P(x_i)P(y_j)}{P(x_i, y_j)} \leq 0$$

Because this expression is of the form

$$\sum_{i=1}^M P_i \log_2 \left( \frac{Q_i}{P_i} \right) \leq 0$$

The above expression can be applied due to the factor  $P(x_i)P(y_j)$ , which is the product of two probabilities, so that it behaves as the quantity  $Q_i$ , which in this expression is a dummy variable that fits the condition  $\sum_i Q_i \leq 1$ . It can be concluded that the average mutual information is a non-negative number. It can also be equal to zero, when the input and the output are independent of each other. A related entropy called the joint entropy is defined as

$$\begin{aligned} H(X, Y) &= \sum_{i,j} P(x_i, y_j) \log_2 \frac{1}{P(x_i, y_j)} \\ &= \sum_{i,j} P(x_i, y_j) \log_2 \frac{P(x_i)P(y_j)}{P(x_i, y_j)} \\ &+ \sum_{i,j} P(x_i, y_j) \log_2 \frac{1}{P(x_i)P(y_j)} \end{aligned}$$

**Theorem 1.5:** Entropies of the binary erasure channel (BEC) The BEC is defined with an alphabet of two inputs and three outputs, with symbol probabilities.

$P(x_1) = \alpha$  and  $P(x_2) = 1 - \alpha$ , and transition probabilities

$$P(y_3/x_2) = 1 - p \text{ and } P(y_2/x_1) = 0,$$

$$\text{and } P(y_3/x_1) = 0$$

$$\text{and } P(y_1/x_2) = p$$

$$\text{and } P(y_2/x_2) = 1 - p$$

**Lemma 1.7.** Given an arbitrary restricted time-discrete, amplitude-continuous channel whose

restrictions are determined by sets  $F_n$  and whose density functions exhibit no dependence on the state  $s$ , let  $n$  be a fixed positive integer, and  $p(x)$  an arbitrary probability density function on Euclidean  $n$ -space.  $p(y|x)$  for the density  $P_n(y_1, \dots, y_n | x_1, \dots, x_n)$  and  $F$  for  $F_n$ . For any real number  $a$ , let

$$A = \left\{ (x, y) : \log \frac{p(y|x)}{p(y)} > a \right\} \quad (1)$$

Then for each positive integer  $u$ , there is a code  $(u, n, \lambda)$  such that

$$\lambda \leq ue^{-a} + P\{(X, Y) \notin A\} + P\{X \notin F\} \quad (2)$$

Where

$$P\{(X, Y) \in A\} = \int_A \dots \int p(x, y) dx dy, \quad p(x, y) = p(x)p(y|x)$$

and

$$P\{X \in F\} = \int_F \dots \int p(x) dx$$

**Proof:** A sequence  $x^{(1)} \in F$  such that

$$P\{Y \in A_{x^{(1)}} | X = x^{(1)}\} \geq 1 - \varepsilon$$

where  $A_x = \{y : (x, y) \in A\}$ ;

Choose the decoding set  $B_1$  to be  $A_{x^{(1)}}$ . Having chosen  $x^{(1)}, \dots, x^{(k-1)}$  and  $B_1, \dots, B_{k-1}$ , select  $x^k \in F$  such that

$$P\left\{Y \in A_{x^{(k)}} - \bigcup_{i=1}^{k-1} B_i | X = x^{(k)}\right\} \geq 1 - \varepsilon;$$

Set  $B_k = A_{x^{(k)}} - \bigcup_{i=1}^{k-1} B_i$ . If the process does not terminate in a finite number of steps, then the sequences  $x^{(i)}$  and decoding sets  $B_i, i = 1, 2, \dots, u$ , form the desired code. Thus assume that the process terminates after  $t$  steps. (Conceivably  $t = 0$ ). We will show  $t \geq u$  by showing that

$\varepsilon \leq te^{-a} + P\{(X, Y) \notin A\} + P\{X \notin F\}$ . We proceed as follows.

Let

$B = \bigcup_{j=1}^t B_j$ . (If  $t=0$ , take  $B = \phi$ ). Then

$$P\{(X,Y) \in A\} = \int_{(x,y) \in A} p(x,y) dx dy$$

$$= \int_x p(x) \int_{y \in A_x} p(y|x) dy dx$$

$$= \int_x p(x) \int_{y \in B \cap A_x} p(y|x) dy dx + \int_x p(x)$$

### C. Algorithms

**Ideals.** Let  $A$  be a ring. Recall that an *ideal*  $a$  in  $A$  is a subset such that  $a$  is a subgroup of  $A$  regarded as a group under addition;

$$a \in a, r \in A \Rightarrow ra \in a$$

The *ideal generated* by a subset  $S$  of  $A$  is the intersection of all ideals  $A$  containing  $S$  ----- it is easy to verify that this is in fact an ideal, and that it consist of all finite sums of the form  $\sum r_i s_i$  with  $r_i \in A, s_i \in S$ . When  $S = \{s_1, \dots, s_m\}$ , we shall write  $(s_1, \dots, s_m)$  for the ideal it generates.

Let  $a$  and  $b$  be ideals in  $A$ . The set  $\{a+b \mid a \in a, b \in b\}$  is an ideal, denoted by  $a+b$ . The ideal generated by  $\{ab \mid a \in a, b \in b\}$  is denoted by  $ab$ . Note that  $ab \subset a \cap b$ . Clearly  $ab$  consists of all finite sums  $\sum a_i b_i$  with  $a_i \in a$  and  $b_i \in b$ , and if  $a = (a_1, \dots, a_m)$  and  $b = (b_1, \dots, b_n)$ , then  $ab = (a_1 b_1, \dots, a_1 b_n, \dots, a_m b_1, \dots, a_m b_n)$ . Let  $a$  be an ideal of  $A$ . The set of cosets of  $a$  in  $A$  forms a ring  $A/a$ , and  $a \mapsto a+a$  is a homomorphism  $\phi: A \mapsto A/a$ . The map  $b \mapsto \phi^{-1}(b)$  is a one to one correspondence between the ideals of  $A/a$  and the ideals of  $A$  containing  $a$ . An ideal  $p$  is *prime* if  $p \neq A$  and  $ab \in p \Rightarrow a \in p$  or  $b \in p$ . Thus  $p$  is prime if and only if  $A/p$  is nonzero and has the property that  $ab=0, b \neq 0 \Rightarrow a=0$ , i.e.,  $A/p$  is an integral domain. An ideal  $m$  is *maximal* if  $m \neq A$  and there does not exist an ideal  $n$  contained strictly between  $m$  and  $A$ . Thus  $m$  is maximal if and only if  $A/m$  has no proper nonzero ideals, and so is a field. Note that  $m$  maximal  $\Rightarrow m$  prime. The ideals of  $A \times B$  are all of the form  $a \times b$ , with  $a$  and  $b$  ideals in  $A$  and  $B$ . To see this, note that if  $c$  is an ideal in  $A \times B$  and

$(a,b) \in c$ , then  $(a,0) = (a,b)(1,0) \in c$  and  $(0,b) = (a,b)(0,1) \in c$ . This shows that  $c = a \times b$  with

$$a = \{a \mid (a,b) \in c \text{ some } b \in b\}$$

and

$$b = \{b \mid (a,b) \in c \text{ some } a \in a\}$$

Let  $A$  be a ring. An  $A$ -algebra is a ring  $B$  together with a homomorphism  $i_B: A \rightarrow B$ . A *homomorphism* of  $A$ -algebra  $B \rightarrow C$  is a homomorphism of rings  $\phi: B \rightarrow C$  such that  $\phi(i_B(a)) = i_C(a)$  for all  $a \in A$ . An  $A$ -algebra  $B$  is said to be *finitely generated* (or of *finite-type* over  $A$ ) if there exist elements  $x_1, \dots, x_n \in B$  such that every element of  $B$  can be expressed as a polynomial in the  $x_i$  with coefficients in  $i(A)$ , i.e., such that the homomorphism  $A[X_1, \dots, X_n] \rightarrow B$  sending  $X_i$  to  $x_i$  is surjective. A ring homomorphism  $A \rightarrow B$  is *finite*, and  $B$  is finitely generated as an  $A$ -module. Let  $k$  be a field, and let  $A$  be a  $k$ -algebra. If  $1 \neq 0$  in  $A$ , then the map  $k \rightarrow A$  is injective, we can identify  $k$  with its image, i.e., we can regard  $k$  as a subring of  $A$ . If  $1=0$  in a ring  $R$ , the  $R$  is the zero ring, i.e.,  $R = \{0\}$ . **Polynomial rings.** Let  $k$  be a field. A *monomial* in  $X_1, \dots, X_n$  is an expression of the form  $X_1^{a_1} \dots X_n^{a_n}$ ,  $a_j \in \mathbb{N}$ . The *total degree* of the monomial is  $\sum a_i$ . We sometimes abbreviate it by  $X^\alpha$ ,  $\alpha = (a_1, \dots, a_n) \in \mathbb{N}^n$ . The elements of the polynomial ring  $k[X_1, \dots, X_n]$  are finite sums  $\sum c_{a_1, \dots, a_n} X_1^{a_1} \dots X_n^{a_n}$ ,  $c_{a_1, \dots, a_n} \in k$ ,  $a_j \in \mathbb{N}$ . With the obvious notions of equality, addition and multiplication. Thus the monomials form a basis for  $k[X_1, \dots, X_n]$  as a  $k$ -vector space. The ring  $k[X_1, \dots, X_n]$  is an integral domain, and the only units in it are the nonzero constant polynomials. A polynomial  $f(X_1, \dots, X_n)$  is *irreducible* if it is nonconstant and has only the obvious factorizations, i.e.,  $f = gh \Rightarrow g$  or  $h$  is constant. **Division in  $k[X]$ .** The division algorithm allows us to divide a nonzero polynomial into another: let  $f$  and  $g$  be

polynomials in  $k[X]$  with  $g \neq 0$ ; then there exist unique polynomials  $q, r \in k[X]$  such that  $f = qg + r$  with either  $r = 0$  or  $\deg r < \deg g$ . Moreover, there is an algorithm for deciding whether  $f \in (g)$ , namely, find  $r$  and check whether it is zero. Moreover, the Euclidean algorithm allows to pass from finite set of generators for an ideal in  $k[X]$  to a single generator by successively replacing each pair of generators with their greatest common divisor. (Pure) **lexicographic ordering** (*lex*). Here monomials are ordered by lexicographic (dictionary) order. More precisely, let  $\alpha = (a_1, \dots, a_n)$  and  $\beta = (b_1, \dots, b_n)$  be two elements of  $\mathbb{N}^n$ ; then  $\alpha > \beta$  and  $X^\alpha > X^\beta$  (lexicographic ordering) if, in the vector difference  $\alpha - \beta \in \mathbb{N}^n$ , the left most nonzero entry is positive. For example,

$XY^2 > Y^3Z^4$ ;  $X^3Y^2Z^4 > X^3Y^2Z$ . Note that this isn't quite how the dictionary would order them: it would put  $XXXYYZZZZ$  after  $XXXYYZ$ . **Graded reverse lexicographic order** (*grevlex*). Here monomials are ordered by total degree, with ties broken by reverse lexicographic ordering. Thus,  $\alpha > \beta$  if  $\sum a_i > \sum b_i$ , or  $\sum a_i = \sum b_i$  and in  $\alpha - \beta$  the right most nonzero entry is negative. For example:

$X^4Y^4Z^7 > X^5Y^5Z^4$  (total degree greater)  
 $XY^5Z^2 > X^4YZ^3$ ,  $X^5YZ > X^4YZ^2$ .

**Orderings on  $k[X_1, \dots, X_n]$ .** Fix an ordering on the monomials in  $k[X_1, \dots, X_n]$ . Then we can write an element  $f$  of  $k[X_1, \dots, X_n]$  in a canonical fashion, by re-ordering its elements in decreasing order. For example, we would write

$f = 4XY^2Z + 4Z^2 - 5X^3 + 7X^2Z^2$   
as  
 $f = -5X^3 + 7X^2Z^2 + 4XY^2Z + 4Z^2$  (*lex*)  
or  
 $f = 4XY^2Z + 7X^2Z^2 - 5X^3 + 4Z^2$  (*grevlex*)

Let  $\sum a_\alpha X^\alpha \in k[X_1, \dots, X_n]$ , in decreasing order:

$$f = a_{\alpha_0} X^{\alpha_0} + a_{\alpha_1} X^{\alpha_1} + \dots, \quad \alpha_0 > \alpha_1 > \dots, \quad \alpha_0 \neq 0$$

Then we define.

- The *multidegree* of  $f$  to be  $\text{multdeg}(f) = \alpha_0$ ;
- The *leading coefficient* of  $f$  to be  $LC(f) = a_{\alpha_0}$ ;
- The *leading monomial* of  $f$  to be  $LM(f) = X^{\alpha_0}$ ;
- The *leading term* of  $f$  to be  $LT(f) = a_{\alpha_0} X^{\alpha_0}$

For the polynomial  $f = 4XY^2Z + \dots$ , the multidegree is (1,2,1), the leading coefficient is 4, the leading monomial is  $XY^2Z$ , and the leading term is  $4XY^2Z$ . **The division algorithm in  $k[X_1, \dots, X_n]$ .** Fix a monomial ordering in  $\mathbb{N}^n$ .

Suppose given a polynomial  $f$  and an ordered set  $(g_1, \dots, g_s)$  of polynomials; the division algorithm then constructs polynomials  $a_1, \dots, a_s$  and  $r$  such that  $f = a_1g_1 + \dots + a_sg_s + r$  Where either  $r = 0$  or no monomial in  $r$  is divisible by any of  $LT(g_1), \dots, LT(g_s)$

**Step 1:** If  $LT(g_1) | LT(f)$ , divide  $g_1$  into  $f$  to get  $f = a_1g_1 + h$ ,  $a_1 = \frac{LT(f)}{LT(g_1)} \in k[X_1, \dots, X_n]$

If  $LT(g_1) \nmid LT(h)$ , repeat the process until  $f = a_1g_1 + f_1$  (different  $a_1$ ) with  $LT(f_1)$  not divisible by  $LT(g_1)$ . Now divide  $g_2$  into  $f_1$ , and so on, until  $f = a_1g_1 + \dots + a_sg_s + r_1$  With  $LT(r_1)$  not divisible by any  $LT(g_1), \dots, LT(g_s)$

**Step 2:** Rewrite  $r_1 = LT(r_1) + r_2$ , and repeat Step 1 with  $r_2$  for  $f$ :  
 $f = a_1g_1 + \dots + a_sg_s + LT(r_1) + r_3$  (different  $a_i$ 's)

**Monomial ideals.** In general, an ideal  $a$  will contain a polynomial without containing the individual terms of the polynomial; for example, the ideal  $a = (Y^2 - X^3)$  contains  $Y^2 - X^3$  but not  $Y^2$  or  $X^3$ .

**DEFINITION 1.5.** An ideal  $a$  is *monomial* if  $\sum c_\alpha X^\alpha \in a \Rightarrow X^\alpha \in a$  all  $\alpha$  with  $c_\alpha \neq 0$ .

**PROPOSITION 1.3.** Let  $a$  be a *monomial ideal*, and let  $A = \{\alpha | X^\alpha \in a\}$ . Then  $A$  satisfies the

condition  $\alpha \in A, \beta \in \square^n \Rightarrow \alpha + \beta \in A$  (\*)  
 And  $a$  is the  $k$ -subspace of  $k[X_1, \dots, X_n]$   
 generated by the  $X^\alpha, \alpha \in A$ . Conversely, if  $A$  is  
 a subset of  $\square^n$  satisfying (\*), then the  $k$ -subspace  
 $a$  of  $k[X_1, \dots, X_n]$  generated by  
 $\{X^\alpha | \alpha \in A\}$  is a monomial ideal.

**PROOF.** It is clear from its definition that a  
 monomial ideal  $a$  is the  $k$ -subspace of  
 $k[X_1, \dots, X_n]$  generated by the set of monomials it contains. If  
 $X^\alpha \in a$  and  $X^\beta \in k[X_1, \dots, X_n]$ .

If a permutation is chosen uniformly and at  
 random from the  $n!$  possible permutations in  $S_n$ ,  
 then the counts  $C_j^{(n)}$  of cycles of length  $j$  are  
 dependent random variables. The joint distribution  
 of  $C^{(n)} = (C_1^{(n)}, \dots, C_n^{(n)})$  follows from Cauchy's  
 formula, and is given by

$$P[C^{(n)} = c] = \frac{1}{n!} N(n, c) = \frac{1}{n!} \left\{ \sum_{j=1}^n j c_j = n \right\} \prod_{j=1}^n \frac{1}{j^{c_j} c_j!}, \quad (1.1)$$

for  $c \in \square_+^n$ .

**Lemma 1.7** For nonnegative integers  
 $m_1, \dots, m_n$ ,

$$E \left( \prod_{j=1}^n (C_j^{(n)})^{m_j} \right) = \left( \prod_{j=1}^n \left( \frac{1}{j} \right)^{m_j} \right) \mathbb{1} \left\{ \sum_{j=1}^n j m_j \leq n \right\} \quad (1.4)$$

*Proof.* This can be established directly by  
 exploiting cancellation of the form  
 $c_j^{[m_j]} / c_j! = 1 / (c_j - m_j)!$  when  $c_j \geq m_j$ , which  
 occurs between the ingredients in Cauchy's formula  
 and the falling factorials in the moments. Write  
 $m = \sum j m_j$ . Then, with the first sum indexed by  
 $c = (c_1, \dots, c_n) \in \square_+^n$  and the last sum indexed by  
 $d = (d_1, \dots, d_n) \in \square_+^n$  via the correspondence  
 $d_j = c_j - m_j$ , we have

$$\begin{aligned} E \left( \prod_{j=1}^n (C_j^{(n)})^{m_j} \right) &= \sum_c P[C^{(n)} = c] \prod_{j=1}^n (c_j)^{m_j} \\ &= \sum_{c: c_j \geq m_j \text{ for all } j} \mathbb{1} \left\{ \sum_{j=1}^n j c_j = n \right\} \prod_{j=1}^n \frac{(c_j)^{m_j}}{j^{c_j} c_j!} \\ &= \prod_{j=1}^n \frac{1}{j^{m_j}} \sum_d \mathbb{1} \left\{ \sum_{j=1}^n j d_j = n - m \right\} \prod_{j=1}^n \frac{1}{j^{d_j} (d_j)!} \end{aligned}$$

This last sum simplifies to the indicator  $\mathbb{1}(m \leq n)$ ,  
 corresponding to the fact that if  $n - m \geq 0$ , then  $d_j = 0$   
 for  $j > n - m$ , and a random permutation in  $S_{n-m}$  must  
 have some cycle structure  $(d_1, \dots, d_{n-m})$ . The moments of

follow immediately as

$$E(C_j^{(n)})^{[r]} = j^{-r} \mathbb{1}\{jr \leq n\} \quad (1.2)$$

We note for future reference that (1.4) can also be  
 written in the form

$$E \left( \prod_{j=1}^n (C_j^{(n)})^{m_j} \right) = E \left( \prod_{j=1}^n Z_j^{m_j} \right) \mathbb{1} \left\{ \sum_{j=1}^n j m_j \leq n \right\}, \quad (1.3)$$

Where the  $Z_j$  are independent Poisson-  
 distribution random variables that satisfy  
 $E(Z_j) = 1/j$

**The marginal distribution of cycle counts** provides  
 a formula for the joint distribution of the cycle  
 counts  $C_j^n$ , we find the distribution of  $C_j^n$  using a  
 combinatorial approach combined with the  
 inclusion-exclusion formula.

**Lemma 1.8.** For  $1 \leq j \leq n$ ,

$$P[C_j^{(n)} = k] = \frac{j^{-k} \sum_{l=0}^{\lfloor n/j \rfloor - k} (-1)^l \binom{\lfloor n/j \rfloor - k}{l}}{k!} \quad (1.1)$$

*Proof.* Consider the set  $I$  of all possible cycles of  
 length  $j$ , formed with elements chosen from  
 $\{1, 2, \dots, n\}$ , so that  $|I| = n^{\lfloor n/j \rfloor}$ . For each  $\alpha \in I$ ,  
 consider the "property"  $G_\alpha$  of having  $\alpha$ ; that is,  
 $G_\alpha$  is the set of permutations  $\pi \in S_n$  such that  $\alpha$   
 is one of the cycles of  $\pi$ . We then have  
 $|G_\alpha| = (n - j)!$ , since the elements of  $\{1, 2, \dots, n\}$   
 not in  $\alpha$  must be permuted among themselves. To  
 use the inclusion-exclusion formula we need to  
 calculate the term  $S_r$ , which is the sum of the  
 probabilities of the  $r$ -fold intersection of properties,  
 summing over all sets of  $r$  distinct properties. There  
 are two cases to consider. If the  $r$  properties are  
 indexed by  $r$  cycles having no elements in common,  
 then the intersection specifies how  $rj$  elements are  
 moved by the permutation, and there are  
 $(n - rj)!\mathbb{1}(rj \leq n)$  permutations in the intersection.

There are  $n^{\lfloor rj \rfloor} / (j^r r!)$  such intersections. For the other case, some two distinct properties name some element in common, so no permutation can have both these properties, and the  $r$ -fold intersection is empty. Thus

$$S_r = (n - rj)! \mathbb{1}(rj \leq n) \\ \times \frac{n^{\lfloor rj \rfloor}}{j^r r! n!} = \mathbb{1}(rj \leq n) \frac{1}{j^r r!}$$

Finally, the inclusion-exclusion series for the number of permutations having exactly  $k$  properties is

$$\sum_{l \geq 0} (-1)^l \binom{k+l}{l} S_{k+l},$$

Which simplifies to (1.1) Returning to the original hat-check problem, we substitute  $j=1$  in (1.1) to obtain the distribution of the number of fixed points of a random permutation. For  $k = 0, 1, \dots, n$ ,

$$P[C_1^{(n)} = k] = \frac{1}{k!} \sum_{l=0}^{n-k} (-1)^l \frac{1}{l!}, \quad (1.2)$$

and the moments of  $C_1^{(n)}$  follow from (1.2) with  $j=1$ . In particular, for  $n \geq 2$ , the mean and variance of  $C_1^{(n)}$  are both equal to 1. The joint distribution of  $(C_1^{(n)}, \dots, C_b^{(n)})$  for any  $1 \leq b \leq n$  has an expression similar to (1.7); this too can be derived by inclusion-exclusion. For any  $c = (c_1, \dots, c_b) \in \mathbb{N}_+^b$  with  $m = \sum c_i$ ,

$$P[(C_1^{(n)}, \dots, C_b^{(n)}) = c] \\ = \left\{ \prod_{i=1}^b \binom{1}{i} \frac{1}{c_i!} \right\} \sum_{\substack{l \geq 0 \text{ with} \\ \sum l_i \leq n-m}} (-1)^{l_1 + \dots + l_b} \prod_{i=1}^b \binom{1}{i} \frac{1}{l_i!} \quad (1.3)$$

The joint moments of the first  $b$  counts  $C_1^{(n)}, \dots, C_b^{(n)}$  can be obtained directly from (1.2) and (1.3) by setting  $m_{b+1} = \dots = m_n = 0$

### The limit distribution of cycle counts

It follows immediately from Lemma 1.2 that for each fixed  $j$ , as  $n \rightarrow \infty$ ,

$$P[C_j^{(n)} = k] \rightarrow \frac{j^{-k}}{k!} e^{-1/j}, \quad k = 0, 1, 2, \dots,$$

So that  $C_j^{(n)}$  converges in distribution to a random variable  $Z_j$  having a Poisson distribution with mean  $1/j$ ; we use the notation  $C_j^{(n)} \rightarrow_d Z_j$  where  $Z_j \square P_o(1/j)$  to describe this. Infact, the limit random variables are independent.

**Theorem 1.6** The process of cycle counts converges in distribution to a Poisson process of  $\square$  with intensity  $j^{-1}$ . That is, as  $n \rightarrow \infty$ ,

$$(C_1^{(n)}, C_2^{(n)}, \dots) \rightarrow_d (Z_1, Z_2, \dots) \quad (1.1)$$

Where the  $Z_j, j = 1, 2, \dots,$  are independent Poisson-distributed random variables with  $E(Z_j) = \frac{1}{j}$

**Proof.** To establish the converges in distribution one shows that for each fixed  $b \geq 1$ , as  $n \rightarrow \infty$ ,  $P[(C_1^{(n)}, \dots, C_b^{(n)}) = c] \rightarrow P[(Z_1, \dots, Z_b) = c]$

### Error rates

The proof of Theorem says nothing about the rate of convergence. Elementary analysis can be used to estimate this rate when  $b=1$ . Using properties of alternating series with decreasing terms, for  $k = 0, 1, \dots, n$ ,

$$\frac{1}{k!} \left( \frac{1}{(n-k+1)!} - \frac{1}{(n-k+2)!} \right) \leq |P[C_1^{(n)} = k] - P[Z_1 = k]| \\ \leq \frac{1}{k!(n-k+1)!}$$

It follows that

$$\frac{2^{n+1}}{(n+1)! n+2} \leq \sum_{k=0}^n |P[C_1^{(n)} = k] - P[Z_1 = k]| \leq \frac{2^{n+1} - 1}{(n+1)!} \quad (1.11)$$

Since

$$P[Z_1 > n] = \frac{e^{-1}}{(n+1)!} \left( 1 + \frac{1}{n+2} + \frac{1}{(n+2)(n+3)} + \dots \right) < \frac{1}{(n+1)!}$$

We see from (1.11) that the total variation distance between the distribution  $L(C_1^{(n)})$  of  $C_1^{(n)}$  and the distribution  $L(Z_1)$  of  $Z_1$

Establish the asymptotics of  $P[A_n(C^{(n)})]$  under conditions  $(A_0)$  and  $(B_{01})$ , where

$$A_n(C^{(n)}) = \bigcap_{1 \leq i \leq n} \bigcap_{r_i+1 \leq j \leq r_i} \{C_{ij}^{(n)} = 0\},$$

and  $\zeta_i = (r_i' / r_{id}) - 1 = O(i^{-g'})$  as  $i \rightarrow \infty$ , for some  $g' > 0$ . We start with the expression

$$P[A_n(C^{(n)})] = \frac{P[T_{0m}(Z') = n]}{P[T_{0m}(Z) = n]} \\ \prod_{\substack{1 \leq i \leq n \\ r_i+1 \leq j \leq r_i}} \left\{ 1 - \frac{\theta}{ir_i} (1 + E_{i0}) \right\} \quad (1.1)$$

$$P[T_{0n}(Z') = n] = \frac{\theta d}{n} \exp \left\{ \sum_{i \geq 1} [\log(1 + i^{-1} \theta d) - i^{-1} \theta d] \right\} \left\{ 1 + O(n^{-1} \phi_{\{1,2,7\}}^*(n)) \right\} \quad (1.2)$$

and

$$P[T_{0n}(Z) = n] = \frac{\theta d}{n} \exp \left\{ \sum_{i \geq 1} [\log(1 + i^{-1} \theta d) - i^{-1} \theta d] \right\} \left\{ 1 + O(n^{-1} \phi_{\{1,2,7\}}^*(n)) \right\} \quad (1.3)$$

Where  $\phi_{\{1,2,7\}}^*(n)$  refers to the quantity derived from  $Z'$ . It thus follows that  $P[A_n(C^{(n)})] \square Kn^{-\theta(1-d)}$  for a constant  $K$ , depending on  $Z$  and the  $r_i$  and computable explicitly from (1.1) – (1.3), if Conditions  $(A_0)$  and  $(B_{01})$  are satisfied and if  $\zeta_i^* = O(i^{-g'})$  from some  $g' > 0$ , since, under these circumstances, both  $n^{-1} \phi_{\{1,2,7\}}^*(n)$  and  $n^{-1} \phi_{\{1,2,7\}}^*(n)$  tend to zero as  $n \rightarrow \infty$ . In particular, for polynomials and square free polynomials, the relative error in this asymptotic approximation is of order  $n^{-1}$  if  $g' > 1$ .

For  $0 \leq b \leq n/8$  and  $n \geq n_0$ , with  $n_0$

$$d_{TV}(L(C[1,b]), L(Z[1,b])) \leq d_{TV}(L(C[1,b]), L(Z[1,b])) \leq \varepsilon_{\{7,7\}}(n,b),$$

Where  $\varepsilon_{\{7,7\}}(n,b) = O(b/n)$  under Conditions  $(A_0), (D_1)$  and  $(B_{11})$ . Since, by the Conditioning Relation,

$$L(C[1,b] | T_{0b}(C) = l) = L(Z[1,b] | T_{0b}(Z) = l),$$

It follows by direct calculation that

$$d_{TV}(L(C[1,b]), L(Z[1,b])) = d_{TV}(L(T_{0b}(C)), L(T_{0b}(Z))) = \max_A \sum_{r \in A} P[T_{0b}(Z) = r] \left\{ 1 - \frac{P[T_{bn}(Z) = n-r]}{P[T_{0n}(Z) = n]} \right\} \quad (1.4)$$

Suppressing the argument  $Z$  from now on, we thus obtain

$$d_{TV}(L(C[1,b]), L(Z[1,b])) = \sum_{r \geq 0} P[T_{0b} = r] \left\{ 1 - \frac{P[T_{bn} = n-r]}{P[T_{0n} = n]} \right\} + \leq \sum_{r > n/2} P[T_{0b} = r] + \sum_{r=0}^{[n/2]} \frac{P[T_{0b} = r]}{P[T_{0b} = n]} \times \left\{ \sum_{s=0}^n P[T_{0b} = s] (P[T_{bn} = n-s] - P[T_{bn} = n-r]) \right\} + \leq \sum_{r > n/2} P[T_{0b} = r] + \sum_{r=0}^{[n/2]} P[T_{0b} = r] \times \sum_{s=0}^{[n/2]} P[T_{0b} = s] \frac{\{P[T_{bn} = n-s] - P[T_{bn} = n-r]\}}{P[T_{0n} = n]} + \sum_{s=0}^{[n/2]} P[T_{0b} = r] \sum_{s=[n/2]+1}^n P[T = s] P[T_{bn} = n-s] / P[T_{0n} = n]$$

The first sum is at most  $2n^{-1}ET_{0b}$ ; the third is bound by

$$\left( \max_{n/2 < s \leq n} P[T_{0b} = s] \right) / P[T_{0n} = n] \leq \frac{2\varepsilon_{\{10.5(1)\}}(n/2, b)}{n} \frac{3n}{\theta P_\theta[0,1]}, \frac{3n}{\theta P_\theta[0,1]} 4n^{-2} \phi_{\{10.8\}}^*(n) \sum_{r=0}^{[n/2]} P[T_{0b} = r] \sum_{s=0}^{[n/2]} P[T_{0b} = s] \frac{1}{2} |r-s| \leq \frac{12\phi_{\{10.8\}}^*(n)}{\theta P_\theta[0,1]} \frac{ET_{0b}}{n}$$

Hence we may take

$$\varepsilon_{\{7,7\}}(n,b) = 2n^{-1}ET_{0b}(Z) \left\{ 1 + \frac{6\phi_{\{10.8\}}^*(n)}{\theta P_\theta[0,1]} \right\} P + \frac{6}{\theta P_\theta[0,1]} \varepsilon_{\{10.5(1)\}}(n/2, b) \quad (1.5)$$

Required order under Conditions  $(A_0), (D_1)$  and  $(B_{11})$ , if  $S(\infty) < \infty$ . If not,  $\phi_{\{10.8\}}^*(n)$  can be replaced by  $\phi_{\{10.11\}}^*(n)$  in the above, which has the required order, without the restriction on the  $r_i$  implied by  $S(\infty) < \infty$ . Examining the Conditions  $(A_0), (D_1)$  and  $(B_{11})$ , it is perhaps surprising to find that  $(B_{11})$  is required instead of just  $(B_{01})$ ; that is, that we should need  $\sum_{i \geq 2} l\varepsilon_{il} = O(i^{-a_1})$  to hold for some  $a_1 > 1$ . A first observation is that a similar problem arises with the rate of decay of  $\varepsilon_{i1}$  as well. For this reason,  $n_1$

is replaced by  $n_1$ . This makes it possible to replace condition  $(A_1)$  by the weaker pair of conditions  $(A_0)$  and  $(D_1)$  in the eventual assumptions needed for  $\varepsilon_{\{7,7\}}(n, b)$  to be of order  $O(b/n)$ ; the decay rate requirement of order  $i^{-1-\gamma}$  is shifted from  $\varepsilon_{i1}$  itself to its first difference. This is needed to obtain the right approximation error for the random mappings example. However, since all the classical applications make far more stringent assumptions about the  $\varepsilon_{i1}, l \geq 2$ , than are made in  $(B_{11})$ . The critical point of the proof is seen where the initial estimate of the difference  $P[T_{bn}^{(m)} = s] - P[T_{bn}^{(m)} = s + 1]$ . The factor  $\varepsilon_{\{10,10\}}(n)$ , which should be small, contains a far tail element from  $n_1$  of the form  $\phi_1^\theta(n) + u_1^*(n)$ , which is only small if  $a_1 > 1$ , being otherwise of order  $O(n^{-a_1+\delta})$  for any  $\delta > 0$ , since  $a_2 > 1$  is in any case assumed. For  $s \geq n/2$ , this gives rise to a contribution of order  $O(n^{-a_1+\delta})$  in the estimate of the difference  $P[T_{bn} = s] - P[T_{bn} = s + 1]$ , which, in the remainder of the proof, is translated into a contribution of order  $O(n^{-a_1+\delta})$  for differences of the form  $P[T_{bn} = s] - P[T_{bn} = s + 1]$ , finally leading to a contribution of order  $bn^{-a_1+\delta}$  for any  $\delta > 0$  in  $\varepsilon_{\{7,7\}}(n, b)$ . Some improvement would seem to be possible, defining the function  $g$  by  $g(w) = 1_{\{w=s\}} - 1_{\{w=s+t\}}$ , differences that are of the form  $P[T_{bn} = s] - P[T_{bn} = s + t]$  can be directly estimated, at a cost of only a single contribution of the form  $\phi_1^\theta(n) + u_1^*(n)$ . Then, iterating the cycle, in which one estimate of a difference in point probabilities is improved to an estimate of smaller order, a bound of the form  $|P[T_{bn} = s] - P[T_{bn} = s + t]| = O(n^{-2}t + n^{-1-a_1+\delta})$  for any  $\delta > 0$  could perhaps be attained, leading to a final error estimate in order  $O(bn^{-1} + n^{-a_1+\delta})$  for any  $\delta > 0$ , to replace  $\varepsilon_{\{7,7\}}(n, b)$ . This would be of the ideal order  $O(b/n)$  for large enough  $b$ , but would still be coarser for small  $b$ .

With  $b$  and  $n$  as in the previous section, we wish to show that

$$\left| d_{TV}(L(C[1, b]), L(Z[1, b])) - \frac{1}{2}(n+1)^{-1} |1 - \theta| E|T_{0b} - ET_{0b}| \right| \leq \varepsilon_{\{7,8\}}(n, b),$$

Where

$\varepsilon_{\{7,8\}}(n, b) = O(n^{-1}b[n^{-1}b + n^{-\beta_{12}+\delta}])$  for any  $\delta > 0$  under Conditions  $(A_0), (D_1)$  and  $(B_{12})$ , with  $\beta_{12}$ . The proof uses sharper estimates. As before, we begin with the formula

$$d_{TV}(L(C[1, b]), L(Z[1, b])) = \sum_{r \geq 0} P[T_{0b} = r] \left\{ 1 - \frac{P[T_{bn} = n - r]}{P[T_{0n} = n]} \right\}_+$$

Now we observe that

$$\left| \sum_{r \geq 0} P[T_{0b} = r] \left\{ 1 - \frac{P[T_{bn} = n - r]}{P[T_{0n} = n]} \right\}_+ - \sum_{r=0}^{\lfloor n/2 \rfloor} \frac{P[T_{0b} = r]}{P[T_{0n} = n]} \right| \times \left| \sum_{s=\lfloor n/2 \rfloor + 1}^n P[T_{0b} = s] (P[T_{bn} = n - s] - P[T_{bn} = n - r]) \right| \leq 4n^{-2} ET_{0b}^2 + (\max_{n/2 < s \leq n} P[T_{0b} = s]) / P[T_{0n} = n] + P[T_{0b} > n/2] \leq 8n^{-2} ET_{0b}^2 + \frac{3\varepsilon_{\{10,5(2)\}}(n/2, b)}{\theta P_\theta[0, 1]}, \quad (1.1)$$

We have

$$\left| \sum_{r=0}^{\lfloor n/2 \rfloor} \frac{P[T_{0b} = r]}{P[T_{0n} = n]} \times \left\{ \sum_{s=0}^{\lfloor n/2 \rfloor} P[T_{0b} = s] (P[T_{bn} = n - s] - P[T_{bn} = n - r]) \right\}_+ - \left\{ \sum_{s=0}^{\lfloor n/2 \rfloor} P[T_{0b} = s] \frac{(s-r)(1-\theta)}{n+1} P[T_{0n} = n] \right\}_+ \right| \leq \frac{1}{n^2 P[T_{0n} = n]} \sum_{r \geq 0} P[T_{0b} = r] \sum_{s \geq 0} P[T_{0b} = s] |s - r| \times \left\{ \varepsilon_{\{10,14\}}(n, b) + 2(r \vee s) |1 - \theta| n^{-1} \left\{ K_0 \theta + 4\phi_{\{10,8\}}^*(n) \right\} \right\} \leq \frac{6}{\theta n P_\theta[0, 1]} ET_{0b} \varepsilon_{\{10,14\}}(n, b) + 4 |1 - \theta| n^{-2} ET_{0b}^2 \left\{ K_0 \theta + 4\phi_{\{10,8\}}^*(n) \right\} \left( \frac{3}{\theta n P_\theta[0, 1]} \right), \quad (1.2)$$

The approximation in (1.2) is further simplified by noting that

$$\sum_{r=0}^{\lfloor n/2 \rfloor} P[T_{0b} = r] \left\{ \sum_{s=0}^{\lfloor n/2 \rfloor} P[T_{0b} = s] \frac{(s-r)(1-\theta)}{n+1} \right\}_+ - \left\{ \sum_{s=0} P[T_{0b} = s] \frac{(s-r)(1-\theta)}{n+1} \right\}_+ \leq \sum_{r=0}^{\lfloor n/2 \rfloor} P[T_{0b} = r] \sum_{s>\lfloor n/2 \rfloor} P[T_{0b} = s] \frac{(s-r)|1-\theta|}{n+1} \leq |1-\theta| n^{-1} E(T_{0b} 1_{\{T_{0b} > n/2\}}) \leq 2|1-\theta| n^{-2} E T_{0b}^2, \quad (1.3)$$

and then by observing that

$$\sum_{r>\lfloor n/2 \rfloor} P[T_{0b} = r] \left\{ \sum_{s \geq 0} P[T_{0b} = s] \frac{(s-r)(1-\theta)}{n+1} \right\} \leq n^{-1} |1-\theta| (E T_{0b} P[T_{0b} > n/2] + E(T_{0b} 1_{\{T_{0b} > n/2\}})) \leq 4|1-\theta| n^{-2} E T_{0b}^2 \quad (1.4)$$

Combining the contributions of (1.2)–(1.3), we thus find  
tha

$$\left| d_{TV}(L(C[1,b]), L(Z[1,b])) \right| - (n+1)^{-1} \sum_{r \geq 0} P[T_{0b} = r] \left\{ \sum_{s \geq 0} P[T_{0b} = s] (s-r)(1-\theta) \right\}_+ \leq \varepsilon_{\{7,8\}}(n,b) = \frac{3}{\theta P_\theta[0,1]} \left\{ \varepsilon_{\{10,5(2)\}}(n/2,b) + 2n^{-1} E T_{0b} \varepsilon_{\{10,14\}}(n,b) \right\} + 2n^{-2} E T_{0b}^2 \left\{ 4 + 3|1-\theta| + \frac{24|1-\theta| \phi_{\{10,8\}}^*(n)}{\theta P_\theta[0,1]} \right\} \quad (1.5)$$

The quantity  $\varepsilon_{\{7,8\}}(n,b)$  is seen to be of the order claimed under Conditions  $(A_0), (D_1)$  and  $(B_{12})$ , provided that  $S(\infty) < \infty$ ; this supplementary condition can be removed if  $\phi_{\{10,8\}}^*(n)$  is replaced by  $\phi_{\{10,11\}}^*(n)$  in the definition of  $\varepsilon_{\{7,8\}}(n,b)$ , has the required order without the restriction on the  $r_i$  implied by assuming that  $S(\infty) < \infty$ . Finally, a direct calculation now shows that

$$\sum_{r \geq 0} P[T_{0b} = r] \left\{ \sum_{s \geq 0} P[T_{0b} = s] (s-r)(1-\theta) \right\}_+ = \frac{1}{2} |1-\theta| E |T_{0b} - E T_{0b}|$$

**Example 1.0.** Consider the point  $O = (0, \dots, 0) \in \square^n$ . For an arbitrary vector  $r$ , the coordinates of the point  $x = O + r$  are equal to the respective coordinates of the vector  $r: x = (x^1, \dots, x^n)$  and  $r = (x^1, \dots, x^n)$ . The vector

$r$  such as in the example is called the position vector or the radius vector of the point  $x$ . (Or, in greater detail:  $r$  is the radius-vector of  $x$  w.r.t an origin  $O$ ). Points are frequently specified by their radius-vectors. This presupposes the choice of  $O$  as the “standard origin”. Let us summarize. We have considered  $\square^n$  and interpreted its elements in two ways: as points and as vectors. Hence we may say that we leading with the two copies of  $\square^n: \square^n = \{\text{points}\}, \square^n = \{\text{vectors}\}$

Operations with vectors: multiplication by a number, addition. Operations with points and vectors: adding a vector to a point (giving a point), subtracting two points (giving a vector).  $\square^n$  treated in this way is called an *n-dimensional affine space*. (An “abstract” affine space is a pair of sets, the set of points and the set of vectors so that the operations as above are defined axiomatically). Notice that vectors in an affine space are also known as “free vectors”. Intuitively, they are not fixed at points and “float freely” in space. From  $\square^n$  considered as an affine space we can precede in two opposite directions:

$\square^n$  as an Euclidean space  $\Leftarrow \square^n$  as an affine space  $\Rightarrow \square^n$  as a manifold. Going to the left means introducing some extra structure which will make the geometry richer. Going to the right means forgetting about part of the affine structure; going further in this direction will lead us to the so-called “smooth (or differentiable) manifolds”. The theory of differential forms does not require any extra geometry. So our natural direction is to the right. The Euclidean structure, however, is useful for examples and applications. So let us say a few words about it:

**Remark 1.0.** *Euclidean geometry.* In  $\square^n$  considered as an affine space we can already do a good deal of geometry. For example, we can consider lines and planes, and quadric surfaces like an ellipsoid. However, we cannot discuss such things as “lengths”, “angles” or “areas” and “volumes”. To be able to do so, we have to introduce some more definitions, making  $\square^n$  a Euclidean space. Namely, we define the length of a vector  $a = (a^1, \dots, a^n)$  to be

$$|a| := \sqrt{(a^1)^2 + \dots + (a^n)^2} \quad (1)$$

After that we can also define distances between points as follows:

$$d(A, B) := |\overline{AB}| \quad (2)$$

One can check that the distance so defined possesses natural properties that we expect: is it always non-negative and equals zero only for coinciding points; the distance from A to B is the same as that from B to A (symmetry); also, for three points, A, B and C, we have

$d(A, B) \leq d(A, C) + d(C, B)$  (the “triangle inequality”). To define angles, we first introduce the scalar product of two vectors

$$(a, b) := a^1 b^1 + \dots + a^n b^n \quad (3)$$

Thus  $|a| = \sqrt{(a, a)}$ . The scalar product is also denote by dot:  $a \cdot b = (a, b)$ , and hence is often referred to as the “dot product”. Now, for nonzero vectors, we define the angle between them by the equality

$$\cos \alpha := \frac{(a, b)}{|a||b|} \quad (4)$$

The angle itself is defined up to an integral multiple of  $2\pi$ . For this definition to be consistent we have to ensure that the r.h.s. of (4) does not exceed 1 by the absolute value. This follows from the inequality

$$(a, b)^2 \leq |a|^2 |b|^2 \quad (5)$$

known as the Cauchy–Bunyakovsky–Schwarz inequality (various combinations of these three names are applied in different books). One of the ways of proving (5) is to consider the scalar square of the linear combination  $a + tb$ , where  $t \in \mathbb{R}$ . As  $(a + tb, a + tb) \geq 0$  is a quadratic polynomial in  $t$  which is never negative, its discriminant must be less or equal zero. Writing this explicitly yields (5). The triangle inequality for distances also follows from the inequality (5).

**Example 1.1.** Consider the function  $f(x) = x^i$  (the  $i$ -th coordinate). The linear function  $dx^i$  (the differential of  $x^i$ ) applied to an arbitrary vector  $h$  is simply  $h^i$ . From these examples follows that we can rewrite  $df$  as

$$df = \frac{\partial f}{\partial x^1} dx^1 + \dots + \frac{\partial f}{\partial x^n} dx^n, \quad (1)$$

which is the standard form. Once again: the partial derivatives in (1) are just the coefficients (depending on  $x$ );  $dx^1, dx^2, \dots$  are linear functions giving on an arbitrary vector  $h$  its coordinates  $h^1, h^2, \dots$ , respectively. Hence

$$df(x)(h) = \partial_{hf(x)} = \frac{\partial f}{\partial x^1} h^1 + \dots + \frac{\partial f}{\partial x^n} h^n, \quad (2)$$

**Theorem 1.7.** Suppose we have a parametrized curve  $t \mapsto x(t)$  passing through  $x_0 \in \mathbb{R}^n$  at  $t = t_0$  and with the velocity vector  $x'(t_0) = v$ . Then  $\frac{df(x(t))}{dt}(t_0) = \partial_v f(x_0) = df(x_0)(v)$  (1)

*Proof.* Indeed, consider a small increment of the parameter  $t : t_0 \mapsto t_0 + \Delta t$ , Where  $\Delta t \mapsto 0$ . On the other hand, we have  $f(x_0 + h) - f(x_0) = df(x_0)(h) + \beta(h)|h|$  for an arbitrary vector  $h$ , where  $\beta(h) \rightarrow 0$  when  $h \rightarrow 0$ . Combining it together, for the increment of  $f(x(t))$  we obtain

$$\begin{aligned} f(x(t_0 + \Delta t)) - f(x_0) &= df(x_0)(v \cdot \Delta t + \alpha(\Delta t) \Delta t) \\ &+ \beta(v \cdot \Delta t + \alpha(\Delta t) \Delta t) \cdot |v \Delta t + \alpha(\Delta t) \Delta t| \\ &= df(x_0)(v) \cdot \Delta t + \gamma(\Delta t) \Delta t \end{aligned}$$

For a certain  $\gamma(\Delta t)$  such that  $\gamma(\Delta t) \rightarrow 0$  when  $\Delta t \rightarrow 0$  (we used the linearity of  $df(x_0)$ ). By the definition, this means that the derivative of  $f(x(t))$  at  $t = t_0$  is exactly  $df(x_0)(v)$ . The statement of the theorem can be expressed by a simple formula:

$$\frac{df(x(t))}{dt} = \frac{\partial f}{\partial x^1} x^1 + \dots + \frac{\partial f}{\partial x^n} x^n \quad (2)$$

To calculate the value of  $df$  at a point  $x_0$  on a given vector  $v$  one can take an arbitrary curve passing Through  $x_0$  at  $t_0$  with  $v$  as the velocity vector at  $t_0$  and calculate the usual derivative of  $f(x(t))$  at  $t = t_0$ .

**Theorem 1.8.** For functions  $f, g : U \rightarrow \mathbb{R}, U \subset \mathbb{R}^n$ ,

$$d(f + g) = df + dg \quad (1)$$

$$d(fg) = df \cdot g + f \cdot dg \quad (2)$$

**Proof.** Consider an arbitrary point  $x_0$  and an arbitrary vector  $v$  stretching from it. Let a curve  $x(t)$  be such that  $x(t_0) = x_0$  and  $x'(t_0) = v$ . Hence

$$d(f + g)(x_0)(v) = \frac{d}{dt}(f(x(t)) + g(x(t)))$$

at  $t = t_0$  and

$$d(fg)(x_0)(v) = \frac{d}{dt}(f(x(t))g(x(t)))$$

at  $t = t_0$  Formulae (1) and (2) then immediately follow from the corresponding formulae for the usual derivative. Now, almost without change the theory generalizes to functions taking values in  $\mathbb{R}^m$  instead of  $\mathbb{R}$ . The only difference is that now the differential of a map  $F:U \rightarrow \mathbb{R}^m$  at a point  $x$  will be a linear function taking vectors in  $\mathbb{R}^n$  to vectors in  $\mathbb{R}^m$  (instead of  $\mathbb{R}$ ). For an arbitrary vector  $h \in \mathbb{R}^n$ ,

$$F(x+h) = F(x) + dF(x)(h) + \beta(h)|h| \quad (3)$$

Where  $\beta(h) \rightarrow 0$  when  $h \rightarrow 0$ . We have  $dF = (dF^1, \dots, dF^m)$  and

$$dF = \frac{\partial F}{\partial x^1} dx^1 + \dots + \frac{\partial F}{\partial x^n} dx^n = \begin{pmatrix} \frac{\partial F^1}{\partial x^1} & \dots & \frac{\partial F^1}{\partial x^n} \\ \dots & \dots & \dots \\ \frac{\partial F^m}{\partial x^1} & \dots & \frac{\partial F^m}{\partial x^n} \end{pmatrix} \begin{pmatrix} dx^1 \\ \dots \\ dx^n \end{pmatrix} \quad (4)$$

In this matrix notation we have to write vectors as vector-columns.

**Theorem 1.9.** For an arbitrary parametrized curve  $x(t)$  in  $\mathbb{R}^n$ , the differential of a map  $F:U \rightarrow \mathbb{R}^m$  (where  $U \subset \mathbb{R}^n$ ) maps the velocity vector  $\dot{x}(t)$  to the velocity vector of the curve  $F(x(t))$  in  $\mathbb{R}^m$ :

$$\frac{dF(x(t))}{dt} = dF(x(t))(\dot{x}(t)) \quad (1)$$

**Proof.** By the definition of the velocity vector,

$$x(t+\Delta t) = x(t) + \dot{x}(t)\Delta t + \alpha(\Delta t)\Delta t \quad (2)$$

Where  $\alpha(\Delta t) \rightarrow 0$  when  $\Delta t \rightarrow 0$ . By the definition of the differential,

$$F(x+h) = F(x) + dF(x)(h) + \beta(h)|h| \quad (3)$$

Where  $\beta(h) \rightarrow 0$  when  $h \rightarrow 0$ . we obtain

$$\begin{aligned} F(x(t+\Delta t)) &= F(x(t) + \underbrace{\dot{x}(t)\Delta t + \alpha(\Delta t)\Delta t}_h) \\ &= F(x(t)) + dF(x(t))(\dot{x}(t)\Delta t + \alpha(\Delta t)\Delta t) + \\ &\quad \beta(\dot{x}(t)\Delta t + \alpha(\Delta t)\Delta t)|\dot{x}(t)\Delta t + \alpha(\Delta t)\Delta t| \\ &= F(x(t)) + dF(x(t))(\dot{x}(t)\Delta t) + \gamma(\Delta t)\Delta t \end{aligned}$$

For some  $\gamma(\Delta t) \rightarrow 0$  when  $\Delta t \rightarrow 0$ . This precisely means that  $dF(x(t))\dot{x}(t)$  is the velocity vector of  $F(x(t))$ . As every vector attached to a point can be viewed as the velocity vector of some curve passing through this point, this theorem gives a clear geometric picture of  $dF$  as a linear map on vectors.

**Theorem 1.10** Suppose we have two maps  $F:U \rightarrow V$  and  $G:V \rightarrow W$ , where  $U \subset \mathbb{R}^n, V \subset \mathbb{R}^m, W \subset \mathbb{R}^p$  (open domains). Let  $F:x \mapsto y = F(x)$ . Then the differential of the composite map  $GoF:U \rightarrow W$  is the composition of the differentials of  $F$  and  $G$ :

$$d(GoF)(x) = dG(y)odF(x) \quad (4)$$

**Proof.** We can use the description of the differential. Consider a curve  $x(t)$  in  $\mathbb{R}^n$  with the

velocity vector  $\dot{x}$ . Basically, we need to know to which vector in  $\mathbb{R}^p$  it is taken by  $d(GoF)$ . the curve  $(GoF)(x(t)) = G(F(x(t)))$ . By the same theorem, it equals the image under  $dG$  of the Anycast Flow vector to the curve  $F(x(t))$  in  $\mathbb{R}^m$ . Applying the theorem once again, we see that the velocity vector to the curve  $F(x(t))$  is the image

under  $dF$  of the vector  $\dot{x}(t)$ . Hence

$$d(GoF)(x) = dG(dF(x)) \quad \text{for an arbitrary}$$

vector  $\dot{x}$ .

**Corollary 1.0.** If we denote coordinates in  $\mathbb{R}^n$  by  $(x^1, \dots, x^n)$  and in  $\mathbb{R}^m$  by  $(y^1, \dots, y^m)$ , and write

$$dF = \frac{\partial F}{\partial x^1} dx^1 + \dots + \frac{\partial F}{\partial x^n} dx^n \quad (1)$$

$$dG = \frac{\partial G}{\partial y^1} dy^1 + \dots + \frac{\partial G}{\partial y^m} dy^m, \quad (2)$$

Then the chain rule can be expressed as follows:

$$d(GoF) = \frac{\partial G}{\partial y^1} dF^1 + \dots + \frac{\partial G}{\partial y^m} dF^m, \quad (3)$$

Where  $dF^i$  are taken from (1). In other words, to get  $d(GoF)$  we have to substitute into (2) the expression for  $dy^i = dF^i$  from (3). This can also be expressed by the following matrix formula:

$$d(GoF) = \begin{pmatrix} \frac{\partial G^1}{\partial y^1} & \dots & \frac{\partial G^1}{\partial y^m} \\ \dots & \dots & \dots \\ \frac{\partial G^p}{\partial y^1} & \dots & \frac{\partial G^p}{\partial y^m} \end{pmatrix} \begin{pmatrix} \frac{\partial F^1}{\partial x^1} & \dots & \frac{\partial F^1}{\partial x^n} \\ \dots & \dots & \dots \\ \frac{\partial F^m}{\partial x^1} & \dots & \frac{\partial F^m}{\partial x^n} \end{pmatrix} \begin{pmatrix} dx^1 \\ \dots \\ dx^n \end{pmatrix} \quad (4)$$

i.e., if  $dG$  and  $dF$  are expressed by matrices of partial derivatives, then  $d(GoF)$  is expressed by the product of these matrices. This is often written as

$$\begin{pmatrix} \frac{\partial z^1}{\partial x^1} & \dots & \frac{\partial z^1}{\partial x^n} \\ \dots & \dots & \dots \\ \frac{\partial z^p}{\partial x^1} & \dots & \frac{\partial z^p}{\partial x^n} \end{pmatrix} = \begin{pmatrix} \frac{\partial z^1}{\partial y^1} & \dots & \frac{\partial z^1}{\partial y^m} \\ \dots & \dots & \dots \\ \frac{\partial z^p}{\partial y^1} & \dots & \frac{\partial z^p}{\partial y^m} \end{pmatrix} \begin{pmatrix} \frac{\partial y^1}{\partial x^1} & \dots & \frac{\partial y^1}{\partial x^n} \\ \dots & \dots & \dots \\ \frac{\partial y^m}{\partial x^1} & \dots & \frac{\partial y^m}{\partial x^n} \end{pmatrix}, \quad (5)$$

Or

$$\frac{\partial z^\mu}{\partial x^a} = \sum_{i=1}^m \frac{\partial z^\mu}{\partial y^i} \frac{\partial y^i}{\partial x^a}, \quad (6)$$

Where it is assumed that the dependence of  $y \in \square^m$  on  $x \in \square^n$  is given by the map  $F$ , the dependence of  $z \in \square^p$  on  $y \in \square^m$  is given by the map  $G$ , and the dependence of  $z \in \square^p$  on  $x \in \square^n$  is given by the composition  $GoF$ .

**Definition 1.6.** Consider an open domain  $U \subset \square^n$ . Consider also another copy of  $\square^n$ , denoted for distinction  $\square_y^n$ , with the standard coordinates  $(y^1 \dots y^n)$ . A system of coordinates in the open domain  $U$  is given by a map  $F: V \rightarrow U$ , where

$V \subset \square_y^n$  is an open domain of  $\square_y^n$ , such that the following three conditions are satisfied :

- (1)  $F$  is smooth;
- (2)  $F$  is invertible;
- (3)  $F^{-1}: U \rightarrow V$  is also smooth

The coordinates of a point  $x \in U$  in this system are the standard coordinates of  $F^{-1}(x) \in \square_y^n$

In other words,

$$F: (y^1 \dots, y^n) \mapsto x = x(y^1 \dots, y^n) \quad (1)$$

Here the variables  $(y^1 \dots, y^n)$  are the “new” coordinates of the point  $x$

**Example 1.2.** Consider a curve in  $\square^2$  specified in polar coordinates as

$$x(t): r = r(t), \varphi = \varphi(t) \quad (1)$$

We can simply use the chain rule. The map  $t \mapsto x(t)$  can be considered as the composition of the maps  $t \mapsto (r(t), \varphi(t)), (r, \varphi) \mapsto x(r, \varphi)$ .

Then, by the chain rule, we have

$$\dot{x} = \frac{dx}{dt} = \frac{\partial x}{\partial r} \frac{dr}{dt} + \frac{\partial x}{\partial \varphi} \frac{d\varphi}{dt} = \frac{\partial x}{\partial r} \dot{r} + \frac{\partial x}{\partial \varphi} \dot{\varphi} \quad (2)$$

Here  $\dot{r}$  and  $\dot{\varphi}$  are scalar coefficients depending on  $t$ , whence the partial derivatives  $\frac{\partial x}{\partial r}, \frac{\partial x}{\partial \varphi}$  are

vectors depending on point in  $\square^2$ . We can compare this with the formula in the “standard” coordinates:

$\dot{x} = e_1 \dot{x} + e_2 \dot{y}$ . Consider the vectors

$\frac{\partial x}{\partial r}, \frac{\partial x}{\partial \varphi}$ . Explicitly we have

$$\frac{\partial x}{\partial r} = (\cos \varphi, \sin \varphi) \quad (3)$$

$$\frac{\partial x}{\partial \varphi} = (-r \sin \varphi, r \cos \varphi) \quad (4)$$

From where it follows that these vectors make a basis at all points except for the origin (where  $r = 0$ ). It is instructive to sketch a picture, drawing vectors corresponding to a point as starting from that point. Notice that  $\frac{\partial x}{\partial r}, \frac{\partial x}{\partial \varphi}$  are, respectively, the velocity vectors for the curves  $r \mapsto x(r, \varphi)$  ( $\varphi = \varphi_0$  fixed) and  $\varphi \mapsto x(r, \varphi)$  ( $r = r_0$  fixed). We can conclude that for an arbitrary curve given in polar coordinates

the velocity vector will have components  $(\dot{r}, \dot{\varphi})$  if as a basis we take  $e_r := \frac{\partial x}{\partial r}, e_\varphi := \frac{\partial x}{\partial \varphi}$ :

$$\dot{x} = e_r \dot{r} + e_\varphi \dot{\varphi} \quad (5)$$

A characteristic feature of the basis  $e_r, e_\varphi$  is that it is not “constant” but depends on point. Vectors “stuck to points” when we consider curvilinear coordinates.

**Proposition 1.3.** The velocity vector has the same appearance in all coordinate systems.

**Proof.** Follows directly from the chain rule and the transformation law for the basis  $e_i$ . In particular, the elements of the basis  $e_i = \frac{\partial x}{\partial x^i}$  (originally, a formal notation) can be understood directly as the velocity vectors of the coordinate lines  $x^i \mapsto x(x^1, \dots, x^n)$  (all coordinates but  $x^i$  are fixed). Since we now know how to handle velocities in arbitrary coordinates, the best way to treat the differential of a map  $F: \square^n \rightarrow \square^m$  is by its action on the velocity vectors. By definition, we set

$$dF(x_0): \frac{dx(t)}{dt}(t_0) \mapsto \frac{dF(x(t))}{dt}(t_0) \quad (1)$$

Now  $dF(x_0)$  is a linear map that takes vectors attached to a point  $x_0 \in \square^n$  to vectors attached to the point  $F(x) \in \square^m$

$$dF = \frac{\partial F}{\partial x^1} dx^1 + \dots + \frac{\partial F}{\partial x^n} dx^n$$

$$(e_1, \dots, e_m) \begin{pmatrix} \frac{\partial F^1}{\partial x^1} & \dots & \frac{\partial F^1}{\partial x^n} \\ \dots & \dots & \dots \\ \frac{\partial F^m}{\partial x^1} & \dots & \frac{\partial F^m}{\partial x^n} \end{pmatrix} \begin{pmatrix} dx^1 \\ \dots \\ dx^n \end{pmatrix}, \quad (2)$$

In particular, for the differential of a function we always have

$$df = \frac{\partial f}{\partial x^1} dx^1 + \dots + \frac{\partial f}{\partial x^n} dx^n, \quad (3)$$

Where  $x^i$  are arbitrary coordinates. The form of the differential does not change when we perform a change of coordinates.

**Example 1.3** Consider a 1-form in  $\square^2$  given in the standard coordinates:

$A = -ydx + xdy$  In the polar coordinates we will have  $x = r \cos \varphi, y = r \sin \varphi$ , hence

$$dx = \cos \varphi dr - r \sin \varphi d\varphi$$

$$dy = \sin \varphi dr + r \cos \varphi d\varphi$$

Substituting into  $A$ , we get

$$A = -r \sin \varphi (\cos \varphi dr - r \sin \varphi d\varphi)$$

$$+ r \cos \varphi (\sin \varphi dr + r \cos \varphi d\varphi)$$

$$= r^2 (\sin^2 \varphi + \cos^2 \varphi) d\varphi = r^2 d\varphi$$

Hence  $A = r^2 d\varphi$  is the formula for  $A$  in the polar coordinates. In particular, we see that this is again a 1-form, a linear combination of the differentials of coordinates with functions as coefficients. Secondly, in a more conceptual way, we can define a 1-form in a domain  $U$  as a linear function on vectors at every point of  $U$ :

$$\omega(v) = \omega_1 v^1 + \dots + \omega_n v^n, \quad (1)$$

If  $v = \sum e_i v^i$ , where  $e_i = \frac{\partial x}{\partial x^i}$ . Recall that the differentials of functions were defined as linear functions on vectors (at every point), and

$$dx^i(e_j) = dx^i \left( \frac{\partial x}{\partial x^j} \right) = \delta_j^i \quad (2) \quad \text{at}$$

every point  $x$ .

**Theorem 1.9.** For arbitrary 1-form  $\omega$  and path  $\gamma$ , the integral  $\int_\gamma \omega$  does not change if we change parametrization of  $\gamma$  provide the orientation remains the same.

*Proof:* Consider  $\left\langle \omega(x(t)), \frac{dx}{dt} \right\rangle$  and

$$\left\langle \omega(x(t(t'))), \frac{dx}{dt} \right\rangle \text{ As}$$

$$\left\langle \omega(x(t(t'))), \frac{dx}{dt} \right\rangle = \left\langle \omega(x(t(t'))), \frac{dx}{dt} \right\rangle \cdot \frac{dt}{dt},$$

Let  $p$  be a rational prime and let  $K = \square(\zeta_p)$ . We write  $\zeta$  for  $\zeta_p$  or this section.

Recall that  $K$  has degree  $\varphi(p) = p-1$  over  $\square$ .

We wish to show that  $O_K = \square[\zeta]$ . Note that  $\zeta$  is

a root of  $x^p - 1$ , and thus is an algebraic integer;

since  $O_K$  is a ring we have that  $\square[\zeta] \subseteq O_K$ . We

give a proof without assuming unique factorization of ideals. We begin with some norm and trace

computations. Let  $j$  be an integer. If  $j$  is not

divisible by  $p$ , then  $\zeta^j$  is a primitive  $p^{\text{th}}$  root of

unity, and thus its conjugates are  $\zeta, \zeta^2, \dots, \zeta^{p-1}$ .

Therefore

$$Tr_{K/\mathbb{Q}}(\zeta^j) = \zeta + \zeta^2 + \dots + \zeta^{p-1} = \Phi_p(\zeta) - 1 = -1$$

If  $p$  does divide  $j$ , then  $\zeta^j = 1$ , so it has only the one conjugate 1, and  $Tr_{K/\mathbb{Q}}(\zeta^j) = p - 1$ . By linearity of the trace, we find that

$$\begin{aligned} Tr_{K/\mathbb{Q}}(1 - \zeta) &= Tr_{K/\mathbb{Q}}(1 - \zeta^2) = \dots \\ &= Tr_{K/\mathbb{Q}}(1 - \zeta^{p-1}) = p \end{aligned}$$

We also need to compute the norm of  $1 - \zeta$ . For this, we use the factorization

$$\begin{aligned} x^{p-1} + x^{p-2} + \dots + 1 &= \Phi_p(x) \\ &= (x - \zeta)(x - \zeta^2) \dots (x - \zeta^{p-1}); \end{aligned}$$

Plugging in  $x = 1$  shows that

$$p = (1 - \zeta)(1 - \zeta^2) \dots (1 - \zeta^{p-1})$$

Since the  $(1 - \zeta^j)$  are the conjugates of  $(1 - \zeta)$ , this shows that  $N_{K/\mathbb{Q}}(1 - \zeta) = p$ . The key result for determining the ring of integers  $O_K$  is the following.

**LEMMA 1.9**

$$(1 - \zeta)O_K \cap \mathbb{Z} = p\mathbb{Z}$$

**Proof.** We saw above that  $p$  is a multiple of  $(1 - \zeta)$  in  $O_K$ , so the inclusion  $(1 - \zeta)O_K \cap \mathbb{Z} \supseteq p\mathbb{Z}$  is immediate. Suppose now that the inclusion is strict. Since  $(1 - \zeta)O_K \cap \mathbb{Z}$  is an ideal of  $\mathbb{Z}$  containing  $p\mathbb{Z}$  and  $p\mathbb{Z}$  is a maximal ideal of  $\mathbb{Z}$ , we must have  $(1 - \zeta)O_K \cap \mathbb{Z} = p\mathbb{Z}$ . Thus we can write

$$1 = \alpha(1 - \zeta)$$

For some  $\alpha \in O_K$ . That is,  $1 - \zeta$  is a unit in  $O_K$ .

**COROLLARY 1.1** For any  $\alpha \in O_K$ ,  $Tr_{K/\mathbb{Q}}((1 - \zeta)\alpha) \in p\mathbb{Z}$

**PROOF.** We have

$$\begin{aligned} Tr_{K/\mathbb{Q}}((1 - \zeta)\alpha) &= \sigma_1((1 - \zeta)\alpha) + \dots + \sigma_{p-1}((1 - \zeta)\alpha) \\ &= \sigma_1(1 - \zeta)\sigma_1(\alpha) + \dots + \sigma_{p-1}(1 - \zeta)\sigma_{p-1}(\alpha) \\ &= (1 - \zeta)\sigma_1(\alpha) + \dots + (1 - \zeta^{p-1})\sigma_{p-1}(\alpha) \end{aligned}$$

Where the  $\sigma_i$  are the complex embeddings of  $K$  (which we are really viewing as automorphisms of  $K$ ) with the usual ordering.

Furthermore,  $1 - \zeta^j$  is a multiple of  $1 - \zeta$  in  $O_K$  for every  $j \neq 0$ . Thus

$Tr_{K/\mathbb{Q}}(\alpha(1 - \zeta)) \in (1 - \zeta)O_K$ . Since the trace is also a rational integer.

**PROPOSITION 1.4** Let  $p$  be a prime number and let  $K = \mathbb{Q}(\zeta_p)$  be the  $p^{\text{th}}$  cyclotomic field. Then  $O_K = \mathbb{Z}[\zeta_p] \cong \mathbb{Z}[x]/(\Phi_p(x))$ ; Thus  $1, \zeta_p, \dots, \zeta_p^{p-2}$  is an integral basis for  $O_K$ .

**PROOF.** Let  $\alpha \in O_K$  and write

$$\alpha = a_0 + a_1\zeta + \dots + a_{p-2}\zeta^{p-2} \quad \text{With } a_i \in \mathbb{Z}.$$

Then

$$\begin{aligned} \alpha(1 - \zeta) &= a_0(1 - \zeta) + a_1(\zeta - \zeta^2) + \dots \\ &+ a_{p-2}(\zeta^{p-2} - \zeta^{p-1}) \end{aligned}$$

By the linearity of the trace and our above calculations we find that  $Tr_{K/\mathbb{Q}}(\alpha(1 - \zeta)) = pa_0$

We also have

$Tr_{K/\mathbb{Q}}(\alpha(1 - \zeta)) \in p\mathbb{Z}$ , so  $a_0 \in \mathbb{Z}$ . Next consider the algebraic integer

$(\alpha - a_0)\zeta^{-1} = a_1 + a_2\zeta + \dots + a_{p-2}\zeta^{p-3}$ ; This is an algebraic integer since  $\zeta^{-1} = \zeta^{p-1}$  is. The same argument as above shows that  $a_1 \in \mathbb{Z}$ , and continuing in this way we find that all of the  $a_i$  are in  $\mathbb{Z}$ . This completes the proof.

**Example 1.4** Let  $K = \mathbb{Q}$ , then the local ring  $\mathbb{Z}_{(p)}$  is simply the subring of  $\mathbb{Q}$  of rational numbers with denominator relatively prime to  $p$ . Note that this ring  $\mathbb{Z}_{(p)}$  is not the ring  $\mathbb{Z}_p$  of  $p$ -adic integers; to get  $\mathbb{Z}_p$  one must complete  $\mathbb{Z}_{(p)}$ . The usefulness of  $O_{K,p}$  comes from the fact that it has a particularly simple ideal structure. Let  $a$  be any proper ideal of  $O_{K,p}$  and consider the ideal  $a \cap O_K$  of  $O_K$ . We claim that  $a = (a \cap O_K)O_{K,p}$ ; That is, that  $a$  is generated by the elements of  $a$  in  $a \cap O_K$ . It is clear from the definition of an ideal that  $a \supseteq (a \cap O_K)O_{K,p}$ . To prove the other inclusion, let  $\alpha$  be any element of  $a$ . Then we can write  $\alpha = \beta/\gamma$  where  $\beta \in O_K$  and  $\gamma \notin p$ . In particular,  $\beta \in a$  (since  $\beta/\gamma \in a$  and  $a$  is an ideal), so  $\beta \in O_K$  and

$\gamma \notin p$ . so  $\beta \in a \cap O_K$ . Since  $1/\gamma \in O_{K,p}$ , this implies that  $\alpha = \beta/\gamma \in (a \cap O_K)O_{K,p}$ , as claimed. We can use this fact to determine all of the ideals of  $O_{K,p}$ . Let  $a$  be any ideal of  $O_{K,p}$  and consider the ideal factorization of  $a \cap O_K$  in  $O_K$ . write it as  $a \cap O_K = p^n b$  For some  $n$  and some ideal  $b$ , relatively prime to  $p$ . we claim first that  $bO_{K,p} = O_{K,p}$ . We now find that

$$a = (a \cap O_K)O_{K,p} = p^n b O_{K,p} = p^n O_{K,p}$$

Since  $bO_{K,p} = O_{K,p}$ . Thus every ideal of  $O_{K,p}$  has the form  $p^n O_{K,p}$  for some  $n$ ; it follows immediately that  $O_{K,p}$  is noetherian. It is also now clear that  $p^n O_{K,p}$  is the unique non-zero prime ideal in  $O_{K,p}$ . Furthermore, the inclusion  $O_K \hookrightarrow O_{K,p}/pO_{K,p}$  Since  $pO_{K,p} \cap O_K = p$ , this map is also surjection, since the residue class of  $\alpha/\beta \in O_{K,p}$  (with  $\alpha \in O_K$  and  $\beta \notin p$ ) is the image of  $\alpha\beta^{-1}$  in  $O_{K/p}$ , which makes sense since  $\beta$  is invertible in  $O_{K/p}$ . Thus the map is an isomorphism. In particular, it is now abundantly clear that every non-zero prime ideal of  $O_{K,p}$  is maximal. To show that  $O_{K,p}$  is a Dedekind domain, it remains to show that it is integrally closed in  $K$ . So let  $\gamma \in K$  be a root of a polynomial with coefficients in  $O_{K,p}$ ; write this polynomial as

$$x^m + \frac{\alpha_{m-1}}{\beta_{m-1}} x^{m-1} + \dots + \frac{\alpha_0}{\beta_0} \quad \text{With } \alpha_i \in O_K \text{ and } \beta_i \in O_{K-p}$$

Set  $\beta = \beta_0 \beta_1 \dots \beta_{m-1}$ . Multiplying by  $\beta^m$  we find that  $\beta\gamma$  is the root of a monic polynomial with coefficients in  $O_K$ . Thus  $\beta\gamma \in O_K$ ; since  $\beta \notin p$ , we have  $\beta\gamma/\beta = \gamma \in O_{K,p}$ . Thus  $O_{K,p}$  is integrally close in  $K$ .

**COROLLARY 1.2.** Let  $K$  be a number field of degree  $n$  and let  $\alpha$  be in  $O_K$  then

$$N'_{K/\mathbb{Q}}(\alpha O_K) = |N_{K/\mathbb{Q}}(\alpha)|$$

**PROOF.** We assume a bit more Galois theory than usual for this proof. Assume first that  $K/\mathbb{Q}$  is Galois. Let  $\sigma$  be an element of  $Gal(K/\mathbb{Q})$ . It is

clear that  $\sigma(O_K)/\sigma(\alpha) \cong O_{K/\alpha}$ ; since  $\sigma(O_K) = O_K$ , this shows that  $N'_{K/\mathbb{Q}}(\sigma(\alpha)O_K) = N'_{K/\mathbb{Q}}(\alpha O_K)$ . Taking the product over all  $\sigma \in Gal(K/\mathbb{Q})$ , we have  $N'_{K/\mathbb{Q}}(N_{K/\mathbb{Q}}(\alpha)O_K) = N'_{K/\mathbb{Q}}(\alpha O_K)^n$  Since  $N_{K/\mathbb{Q}}(\alpha)$  is a rational integer and  $O_K$  is a free  $\mathbb{Z}$ -module of rank  $n$ ,

$O_K / N_{K/\mathbb{Q}}(\alpha)O_K$  Will have order  $N_{K/\mathbb{Q}}(\alpha)^n$ ; therefore

$$N'_{K/\mathbb{Q}}(N_{K/\mathbb{Q}}(\alpha)O_K) = N_{K/\mathbb{Q}}(\alpha O_K)^n$$

This completes the proof. In the general case, let  $L$  be the Galois closure of  $K$  and set  $[L:K] = m$ .

Since only a few axial scans at selected radii from the visual axis are needed to determine corneal profile, thickness, and refractive power. Full cross-sectional images of the anterior chamber may be helpful in visualizing intraocular tumors, foreign bodies, or angle-closure glaucoma. OCT tomographs of the crystalline lens have the potential to provide a new imaging technique and objective grading diagnostic for cataracts. OCT may have advantages over other optical methods of cataract evaluation because the relatively low scattering of 830 nm light relative to visible light and the high detection sensitivity of OCT allow image formation even through dense cataracts. For an initial demonstration of cataract evaluation with OCT, a nuclear cataract was induced in a fresh, enucleated bovine eye by immersion in a temperature-controlled saline bath. Cold induces reversible cataractous changes by the accumulation of crystalline droplets in the lens nucleus and the phase separation of proteins in the lens cell cytoplasm [32]. OCT tomographs of the cataract taken at 10, 15, and 20 degrees C, clearly displaying the disappearance of the cataract with increasing temperature. Cataract densities, blindly graded ophthalmoscopically prior to image acquisition, were 4+, 1+, and 0, respectively. The nuclear cataract at 10 degrees C is visible as a bright scatterer of incident light; however, light penetration through the cataract remains sufficient to reveal the posterior capsule and a central decrease in opacity which was not visible ophthalmoscopically until the temperature is raised to 14 degrees. To provide increased sensitivity to weakly backscattered light, the reference mirror scanning velocity is reduced to 39 mm/s and each image is averaged 10 times, resulting in an image acquisition time of 10 min. Although high sensitivity is necessary to detect and quantify the scattering from early-developing cataracts, high resolution and a large image area are probably not necessary for clinical cataract grading. Thus, a substantial reduction in image acquisition

time may be achieved for clinical application. The tomographs in Figs. 8 and 9 provide information on the large scale morphology of anterior segment structures. By narrowing the field of view, one can obtain high resolution images of ocular microstructure in the anterior eye which may provide important histopathological information concerning disease progression and the healing process *in vivo*. To demonstrate the ability of OCT to visualize cellular changes noninvasively in tissue morphology, we used OCT to evaluate the damage and healing due to laser induced photocoagulation of the cornea [33]. Laser thermokeratoplasty (LTK) of the cornea is a relatively new therapy currently under study for the treatment of keratoconus, refractive errors of the eye, and astigmatism [34]. In this technique, laser coagulations are placed in the corneal stroma circumferentially around the visual axis. Thermally induced collagen shrinkage [35] at each coagulation point leads to alterations in the mechanical properties of the cornea and modification of the corneal profile. Figure 10 shows an *in vivo* OCT tomography taken one month post-operatively of a thermal photocoagulation lesion in a rabbit cornea. An Ho:YSGG laser was used to deliver 100 mJ of 2.1  $\mu\text{m}$  light to the cornea through a 200  $\mu\text{m}$  diameter fiber. During post-operative imaging, the rabbit was anesthetized to minimize the effects of ocular motion. The reference mirror scanning velocity was reduced to 39 mm/s to obtain a corresponding factor of four increase in detection sensitivity. In the normal cornea surrounding the lesion, both the stratified corneal epithelium and the single-cell layer endothelium are well visualized in the tomograph as distinct thick and thin layers of high backscattering surrounding the less reflective stroma. The thermally induced collagen shrinkage in the stroma appears as an area of increased reflectivity, clearly demarcating the radial extent and penetration depth of the photocoagulation burn. Several important features of the healing process are also documented. Regrowth and thickening of the epithelium are clearly seen above the lesion, while the endothelial cell layer remains intact and undamaged just below the photocoagulation. The capability of OCT for non-invasive assessment of histopathology *in vivo* may be important in the clinical evaluation and longitudinal study of diverse ocular disease in both humans and animal models. OCT can record both changes at the cellular level and differences in large scale morphology, showing significant promise as a potential adjunct to keratorefractive therapies such as LTK, where both realtime monitoring of the surgery in progress and post-operative follow-up may require evaluation of the gross corneal curvature as well as cell damage and photocoagulation penetration depth. As a research tool, OCT may be also beneficial in determining the LTK exposure parameters that

provide stable alterations in corneal profile without endothelial cell damage.

OCT is a novel biomedical imaging technique that provides cross-sectional tomographs of optical backscattering within tissue with micron scale resolution in all dimensions and extremely high sensitivity. We have developed a fiber-optic implementation of the interferometric system, which uses a compact and inexpensive continuous-wave super-luminescent diode source. The beam-steering galvanometers and fiber-optics are easily retrofitted to standard slit-lamp examination systems, allowing facile alignment and operator visualization of the scanning probe beam in the eye. A real-time display of the tomograph in progress is provided on a computer monitor. Several features make OCT particularly attractive for imaging ocular tissue. OCT has superior resolution to conventional clinical ultrasound, which is limited by the acoustic wavelength in tissue to about 150  $\mu\text{m}$ , and unlike ultrasound does not require contact with or saline immersion of the eye. Non-contact measurements increase patient comfort during examination and are necessary for realtime intra-operative monitoring of corneal refractive surgeries. High-resolution imaging is possible even in the posterior eye, where signal attenuation limits the application of high-frequency ultrasound and ocular optics prevent high depth-resolution in scanning laser confocal imaging systems. In contrast to both SLT and SLO, the axial resolution of OCT only depends on the temporal coherence properties of the source, and not on the pupil-limited numerical aperture of the eye or on ocular aberrations. Micron scale axial resolution is particularly important for the early diagnosis and monitoring of degenerative retinal diseases.

ACUTE myocardial infarction (AMI), most frequently caused by the disruption of a vulnerable atherosclerotic plaque, is the leading cause of death in the western world [10]. Thin-cap fibroatheromas (TCFAs), the predominant form of vulnerable plaques resulting in sudden cardiac death [11], [12], have been defined as plaques with a large lipid pool, a thin fibrous cap ( $<65 \mu\text{m}$ ), and activated macrophages near or within the fibrous cap [13]–[15]. The rupture of a TCFA, which may be precipitated by biomechanical stresses [16], [17], causes the bloodstream to be exposed to procoagulant factors, forming a nidus for thrombus. In some instances, the thrombus can impede blood flow to downstream myocardium, triggering an acute coronary event [18]. In addition to TCFA, coronary artery thrombosis has also been attributed to eroded plaques and superficial calcific nodules [6], [19], [20]. Regardless of the underlying pathologic substrate, we do not understand which plaques will give rise to coronary thrombosis in any given patient. For this reason, current vulnerable plaque research is focused on the detection and study of the natural history of these high-risk lesions. Data

gained from this research will provide clinicians with the information and tools required to guide pharmacologic and/or interventional management. Patients presenting with stenotic coronary lesions may be treated with stent implantation during percutaneous coronary intervention (PCI). The role of stent placement is to restore and maintain blood flow through the artery. While effective for this purpose, in-stent restenosis, caused by aggressive neointimal hyperplasia, is a significant problem with bare metal stents (BMS), leading to the need for a repeat PCI in a substantial number of patients. Drug-eluting stents (DES), coated with an agent designed to attenuate neointimal growth, reduce this problem, but may result in delayed endothelial healing and rare cases of late stent thrombosis [21]–[24]. The potential risk of stent thrombosis in patients with DES mandates long-term administration of anti-clotting drugs, which are expensive and have their own bleeding-associated complications. Given these difficulties encountered with coronary stenting, there is a need for a tool to evaluate the stent healing process, which may be used to tailor antiplatelet regimen durations on an individual patient basis. A number of imaging modalities have been investigated for studying vulnerable plaques in the hope of uncovering new knowledge regarding this disease process [25]. Both conventional and experimental intravascular imaging modalities include intravascular ultrasound (IVUS) [26]–[30], magnetic resonance imaging (MRI) [31]–[33], optical coherence tomography (OCT) [34], [35], angiography [6], [36]–[40], thermography [41], [42] and near-infrared [43], fluorescence [44], [45], and Raman spectroscopy [9], [46], [47]. Of these, OCT is the only imaging modality with sufficient resolution to visualize the majority of the pathologic features currently associated with the vulnerable plaque [4]. OCT has been utilized as an investigative imaging tool for the assessment of coronary artery pathology for a number of years [48], and has additionally been effective in evaluating the effects of coronary stenting [49]. Early OCT imaging studies were performed using systems based on time-domain OCT (TD-OCT) technology [5], [7], [35], [48]–[53]. One difficulty with conducting coronary OCT in vivo is the need to remove blood from the imaging field in order to clearly visualize the artery wall. Methods employed to displace blood during OCT imaging include flushing the artery with saline both with and without proximal balloon occlusion. The prolonged balloon occlusion or flush times necessary for TD-OCT increased the risk of myocardial ischemia during the procedure, and together with the increased procedural complexity, may have limited the widespread clinical adoption of this technology. With extensive training expert users were able to mitigate these risks while acquiring pullbacks of up to 3–5 cm in length [54], [55]. As a result, thousands of coronary patients have been

imaged with OCT at several hundred sites around the world with commercially available TD-OCT systems and over 200 studies have been published, mostly in clinical journals. With the advent of second generation Fourier-domain OCT (FD-OCT), which enables high-quality imaging at speeds up to 100× that of TD-OCT, 3-D imaging of long coronary segments during a brief transparent media flush is now possible. This paper addresses the imaging principles of OCT that make it an ideal tool for interrogating coronary microstructure, in addition to the recent developments in the technology, which have increased the likelihood that this imaging modality will be widely adopted in cardiology.

#### IV. TIME-DOMAIN OPTICAL COHERENCE

Intravascular OCT is a structural imaging modality that is similar in principle to IVUS. With OCT, the echo time delay of the incident light, rather than acoustic waves, is measured using low-coherence interferometry [35]. In TD-OCT systems, a broadband light source is split into two arms, a reference arm and a sample arm. The reference arm light typically illuminates a reflector and the sample arm light is directed toward the coronary wall. Light returned from both arms is then recombined and detected. When the optical path length traveled by the light in each arm is within the coherence length of the source, the cross correlation of the two electromagnetic fields results in an interference pattern, the amplitude of which may be mapped to a pixel intensity value. By scanning the optical delay of the reference arm, interference fringes from discrete locations within the tissue are obtained and may be assembled to form profiles of reflectivity as a function of depth or A-lines. 2-D and 3-D OCT images are obtained by scanning the sample arm beam across the sample and recording A-lines at each scan position. OCT systems are based on fiber-optic technology, and therefore, are highly conducive to catheter-based imaging required for many clinical applications [56]. OCT imaging of the coronary artery

was first demonstrated in early in vitro studies, where investigators described the visualization of coronary microstructure including the adventitia, media, and intima [48], [50]. Image criteria for the differentiation of coronary artery microstructures have been developed and validated in histopathologic correlative imaging studies, conducted on autopsy specimens ex vivo (see Table I). The classification criteria that are currently utilized to interpret lesion morphology in the clinical setting were developed and prospectively tested by Yabushita et al. [7]. In this study, 357 OCT-histology correlated images of atherosclerotic lesions were obtained from 90 cadavers. The investigators found that fibrous plaques could be identified by homogeneous signal-rich regions, fibrocalcific plaques by signal-poor regions with

sharp borders, and lipid-rich plaques by signal-poor regions with diffuse borders (see Fig. 1). The sensitivity and specificity for plaque characterization based on these criteria were reported to range from 71% to 79% and 97% to 98% for fibrous plaques, 95% to 96% and 97% for fibrocalcific plaques, and 90% to 94% and 90% to 92% for lipid-rich plaques [7]. In addition to the discrimination of plaque type, the capability of OCT to identify arterial macrophages has also been reported [2]. This study showed that macrophage density measured by OCT was correlated to immunohistochemical CD68 staining of macrophages from corresponding histopathologic slides ( $r = 0.84, P < 0.0001$ ) [2]. Given that the presence of activated macrophages in the atherosclerotic plaque are thought to increase plaque vulnerability and probability of rupture [57]–[59]. The knowledge of macrophage distribution and density that may be determined by intracoronary OCT, may prove useful for evaluating arterial inflammation and plaque vulnerability. In 1999, the first in vivo intravascular OCT study was performed in the abdominal aorta of New Zealand white rabbits [51]. Using a 2.9 Fr OCT catheter in conjunction with a nonocclusive saline flush, the normal arterial wall microstructure, including the media and adventitia, were identified [51]. Following this initial demonstration, in vivo OCT imaging of coronary arteries was demonstrated in five swine [60]. The study revealed that intravascular OCT images provided superior resolution when compared to IVUS images obtained from the same locations, and enabled the visualization of features, such as the intima, including intimal flaps and defects, disruptions in the media, and stent strut apposition that could not be identified by IVUS [60]. Based on the ability of OCT to discriminate between various intracoronary plaque microstructures and the potential of this imaging modality to have significant clinical impact, the first intravascular clinical studies with TD-OCT were published in 2002, demonstrating the safety and feasibility of this technique [5], [7], [52], [53]. Intracoronary OCT imaging in living patients enabled the visualization of coronary artery walls with unprecedented resolution. As in prior animal studies, in vivo OCT in the clinical setting was found to provide additional, more detailed structural information when compared to corresponding images obtained with IVUS [5]. In the years, since this initial demonstration, OCT has been used extensively by a number of investigators in the clinical realm for assessing coronary plaque features [61]–[65], stent placement [49], [66], [67], apposition [49], [68]–[70], stent strut coverage [71]–[75], and thrombus [76]. While early studies demonstrated a niche for TD-OCT during PCI, the clinical utility of the technology was hampered by relatively low-image acquisition rates (2–4 kHz, A-line rates), which is because of the need for mechanical actuation of the

reference arm, and as a consequence of the inverse relationship between TD-OCT imaging speed and signal-to-noise ratio. The relatively slow image acquisition rates of TD-OCT was problematic as flushing blood from the field of view was the only practical solution to obtaining clear images of the artery wall and the duration of a bolus of saline within the coronary artery was limited to approximately 2 s. The recognition that the combination of a substantial increase in acquisition speed with a short nonocclusive flush could solve the blood problem and enable screening of long coronary segments [77], was the key advance that has taken intracoronary OCT to the next level required for widespread clinical adoption.

## V. OPTICAL FREQUENCY DOMAIN IMAGING

FD-OCT was the critical technical advance that enabled imaging at sufficient speeds for coronary screening during a brief, nonocclusive flush. One form of FD-OCT, optical frequency domain imaging (OFDI) [78] also called swept-source OCT (SS-OCT) [79], is the particular implementation of FD-OCT, used in most state of the art intracoronary OCT systems. With OFDI, the cross correlation of the optical signal returning from the sample and reference arms is sampled as a function of wavenumber rather than time. The spectrally resolved interference between the sample and reference arms is generated using a rapidly tuned wavelength-swept light source with a narrow instantaneous linewidth. A square-law photodetector is used to acquire the interference signal between the two arms, while the optical path-length of the arms remain constant. Each frequency component of the interference signal is associated with a discrete depth location within the tissue. To generate an A-line, the Fourier transform of the interference fringe is calculated [78]. As in TD-OCT, 2-D and 3-D OFDI images are acquired by scanning the light from the sample arm over the tissue. By detecting all depths of the A-line simultaneously during a single sweep of the light source, the detection sensitivity of OFDI is theoretically increased to a maximum of several orders of magnitude over TD-OCT [78], [79]. This increased sensitivity may be leveraged to increase the imaging speed, enabling 3-D imaging of coronary artery segments during a short nonocclusive saline/radiocontrast purge. An additional advantage of OFDI technology is that it is possible to double the interferometric ranging depth by creating a very narrow instantaneous linewidth [80], [81] or by utilizing both the positive and negative differential delays [82]–[86]. This extended ranging depth can be achieved by shifting the frequency of the detector signal by a constant value, using an acoustooptical frequency shifter in the interferometer [86], or by acquiring both the in-

phase (real) and quadrature (imaginary) components of the interferometric signal [82]–[85]. The increased ranging depths (>7 mm) now obtained with OFDI allow imaging of even the largest human coronary arteries [77]. Intracoronary OFDI was first demonstrated in swine studies in vivo in 2006, where comprehensive microscopy of long segments of coronary arteries was presented [77]. Forty-four in vivo swine intracoronary OFDI datasets were acquired at 108 frames per second with pullback speeds of 5 mm/s in segments up to 6 cm in length [77]. The imaging system used in this study had a source tuning range of 111 nm at a 54-kHz A-line rate corresponding to an image acquisition rate of 108 frames per second [77]. The system utilized dual-balanced, polarization diverse detection, in addition to frequency shifting to remove the depth degeneracy providing a ranging depth of 7.3 mm [77], [86]. To highlight the volumetric capabilities of the imaging technology, the investigators performed angioplasty followed by stenting in the circumflex artery of one swine. The corresponding cross-sectional OFDI images revealed clear stent strut visualization in addition to dissected intima and media as a result of the balloon angioplasty [77]. By presenting the volumetric data in 3-D, a greater appreciation of the artery structure was realized. In 2008, the same group translated this technology to the clinical setting and published the first demonstration of intracoronary OFDI in three human patients undergoing PCI [87]. Intracoronary OFDI datasets from three patients were presented, with imaging rates of 100 frames per second, and pullback speeds ranging from 5 to 20 mm/s [87]. Fig. 2 highlights the long arterial segments that can be successfully imaged with OFDI. The imaging core was translated at a speed of 20 mm/s with a frame rate of 100 frames per second (frame size: 1536 axial points  $\times$  512 A-lines) resulting in a longitudinal imaging pitch of 200  $\mu$ m. During image acquisition, the raw data was continuously streamed to a hard disk drive at a rate of 320 MB/s. The volumetric OFDI data of a right coronary artery was obtained during a single limited duration flush at 3 mL/s through a 7 Fr guide catheter. Using these parameters, a 7-cm longitudinal OFDI pullback of clear blood-free imaging was obtained in under 4 s. The single pullback shows a proximal BMS that was placed nine years prior to imaging, and a DES placed immediately prior to imaging. The wealth of information obtained in the single pullback is highlighted by the volumetric renderings. These renderings were created offline by manually segmenting the images according to previously validated image criteria [1], [2], [7], [49] for the identification of the artery wall, lipid pools, calcific nodules, and stent struts. The macrophages highlighted in the renderings were automatically segmented with previously validated normalized image intensity metrics [2], together with the manual

removal of outliers. Each of the segmented features was rendered in a different color according to the following scheme: red = artery wall, yellow = lipid pool, white = calcific nodule, blue = stent, grey = guide wire, and green = macrophage. The individual renderings were then recombined to form the final 3-D image. Due to the manual segmentation process, the time required to construct the final volume renderings approached a couple of hours, however, with the development of automated and semiautomated image processing algorithms, these times may be considerably reduced. The volume renderings clearly show a high degree of tissue coverage on the BMS. In addition, the placement of the DES over a lipid-rich plaque can be visualized. Together with Figs. 2 and 3 highlights the high level of detail that can be observed with OFDI. Of specific interest in coronary intervention are issues relating to the use and effectiveness of stents, particularly stent placement including individual stent strut apposition, and tissue coverage over the struts.

## VI. POLARIZATION SENSITIVE OPTICAL COHERENCE TOMOGRAPHY

Polarization sensitive OCT (PS-OCT), another embodiment of OCT, provides a measure of tissue birefringence by detecting polarization changes in the light returning from the tissue sample being imaged [88], [89]. When light travels through tissues that exhibit form birefringence, orthogonal polarization components of the light will undergo phase retardation with respect to one another. This degree of phase retardation is dependent on the orientation of the polarization state with respect to the organized linear structures within the tissue, such as collagen fibers [90]. The detected birefringence increases in tissues containing highly organized linear structures. PS-OCT provides complementary image information to structural OCT images that may assist in the identification of the intravascular tissue composition, and may additionally provide insight to the mechanical stability of atherosclerotic plaques [91]. PS-OCT has been demonstrated in histopathologic correlative studies conducted ex vivo to provide a quantitative measure of the collagen content, collagen fiber thickness, and smooth muscle content in atherosclerotic plaques [91]. In 2006, using a spectral-domain PS-OCT imaging system, Nadkarni et al. imaged aortic plaques and compared the PS-OCT spatially averaged birefringence with the plaque collagen content and thickness, and smooth muscle cell content measured from histologic sections stained with picrosirius red and alpha-smooth muscle actin, respectively [91]. This ex vivo study revealed a high-positive correlation between the PS-OCT measured birefringence and the total collagen content ( $r = 0.67$ ,  $p = 0.001$ ), the thick

collagen fiber content ( $r = 0.76$ ,  $p = 0.001$ ), and the smooth muscle cell content ( $r = 0.74$ ,  $p = 0.01$ ) [91], providing compelling evidence that the measurement of artery birefringence may aid in determining the tissue composition of plaques, information that may be used to assess mechanical stability. In intravascular and other catheter-based PS-OCT systems, it is necessary to use optical fibers to transmit the imaging signal to the tissue of interest. Maintaining the polarization state of the transmitted light in fiber-based systems is difficult, even with polarization maintaining fibers, as the polarization state is susceptible to stresses acting on the fiber. One method for circumventing this issue is to modulate the polarization state of the source incident on the sample tissue between two perpendicular states in successive A-line pairs. This modulation ensures that the polarization state of the light source differs for at least one of a successive pair of A-lines, from that of the linear birefringence axis of the sample. Each A-line pair is subsequently combined to form a single axial profile, using either Stokes vector [92] or Jones matrix [93] analysis. This method of fiber-based PS-OCT has been demonstrated in both spectraldomain PS-OCT [94]–[96] and OFDI systems [97]. Recently however, a novel approach to PS-OCT unique to OFDI has been demonstrated that utilizes frequency multiplexing to enable illumination and detection of two polarization states simultaneously [98]. This new implementation of PS-OFDI obviates the need for modulating the polarization state of the source between successive A-lines. PS-OFDI with frequency multiplexing has been demonstrated in ex vivo studies through an intracoronary catheter at an A-line rate of 62 kHz [98]. Fig. 4 shows both a structural and a PS-OFDI image acquired from a human coronary artery in vivo. The PS-OFDI image adds additional detail regarding the structural integrity of the artery that can be inferred from the tissue birefringence strength map.

## VII. CONCLUSION

Many of the early challenges faced with the use of intravascular OCT in the clinical setting were overcome with the development of OFDI, the most significant being imaging speed. While the laser and detection electronics are capable of operating faster still, the ability of acquisition electronics and data processing to keep pace, remains a challenge. Recently however, newer acquisition electronic systems have been developed enabling the acquisition and storage of data at rates approaching 1 GB/s. Additionally, solutions to alleviate CPU processing requirements have been implemented, using hardware components, such as digital signal processors (DSPs) [99] and field programmable gate arrays (FPGAs) [100]. These hardware solutions may be integrated into OFDI systems to handle much of the pre and postacquisition processing, thus

enabling both real-time display and an increase in the data transfer rates achievable [99]–[102]. Bit-depth reduction with a minimal associated loss in the signal-to-noise ratio of the OCT images, may also result in an increase in image acquisition rates due to the reduced bandwidth and storage requirements at lower bit-depths [80], [103]. Coupled with the rapidly increasing use of intravascular OCT in catheterization laboratories, is a pressing need for automated and semiautomated image processing techniques for the evaluation of coronary features including classification based on tissue pathology, stent strut identification and quantification of strut tissue coverage. To date the vast majority of this type of evaluation is manually performed by expert intracoronary OCT readers. This process involves an extremely large time commitment and is subject to interobserver variations. In the case of quantitative feature analyses, such as stent strut coverage or lumen diameter analysis, well-defined and validated protocols are required in addition to controlled image processing steps to account for variances in the refractive indexes of both the tissue and flushing media. While preliminary studies have been conducted describing semiautomated analyses of OCT image data [104], the development of appropriate automated and semiautomated image analysis tools could improve the ease of use of intravascular OCT, particularly in nonspecialized catheterization centers that may have little or no intravascular OCT expertise. With the evolution of OFDI and PS-OFDI, there is also an increasing need for improved visual display techniques that can highlight relevant features, provide an enhanced appreciation of the 3-D morphology, and can amalgamate the complementary information into user-friendly maneuverable 3-D displays. In order to fully appreciate the complex 3-D morphology of the artery, investigators are exploring various display techniques ranging from standard longitudinal and transverse cross-sectional displays to intensity-based volume rendering, and more complex methods involving segmentation and pseudocoloring based on tissue characterization with subsequent 3-D volume rendering [87]. While preliminary work has been demonstrated by some investigators in the manipulation, analysis, and display of intravascular OCT datasets, further work in this field is needed, which may be leveraged from the extensive research performed with other imaging modalities, such as IVUS [105], [106]. The potential clinical utility of intravascular OCT has no doubt increased as a direct result of the development of highspeed OFDI technology. OFDI enables imaging of long coronary segments, previously difficult with the first generation TD-OCT, during a brief flush with an optically transparent media. Based on the status of currently available imaging modalities for interrogating the coronary arteries, intravascular OCT is uniquely situated to play a critical role in

improving our understanding of the vulnerable plaque, in addition to possibly guiding patient management and monitoring the response to PCI.

#### A. Authors and Affiliations

Dr Akash Singh is working with IBM Corporation as an IT Architect and has been designing Mission Critical System and Service Solutions; He has published papers in IEEE and other International Conferences and Journals.

He joined IBM in Jul 2003 as a IT Architect which conducts research and design of High Performance Smart Grid Services and Systems and design mission critical architecture for High Performance Computing Platform and Computational Intelligence and High Speed Communication systems. He is a member of IEEE (Institute for Electrical and Electronics Engineers), the AAAI (Association for the Advancement of Artificial Intelligence) and the AACR (American Association for Cancer Research). He is the recipient of numerous awards from World Congress in Computer Science, Computer Engineering and Applied Computing 2010, 2011, and IP Multimedia System 2008 and Billing and Roaming 2008. He is active research in the field of Artificial Intelligence and advancement in Medical Systems. He is in Industry for 18 Years where he performed various role to provide the Leadership in Information Technology and Cutting edge Technology.

#### REFERENCES

- [1] B. D. MacNeill, I. K. Jang, B. E. Bouma, N. Iftimia, M. Takano, H. Yabushita, M. Shishkov, C. R. Kauffman, S. L. Houser, H. T. Aretz, D. DeJoseph, E. F. Halpern, and G. J. Tearney, "Focal and multi-focal plaque macrophage distributions in patients with acute and stable presentations of coronary artery disease," *J. Amer. Coll. Cardiol.*, vol. 44, pp. 972–979, Sep. 1, 2004.
- [2] G. J. Tearney, H. Yabushita, S. L. Houser, H. T. Aretz, I. K. Jang, K. H. Schlendorf, C. R. Kauffman, M. Shishkov, E. F. Halpern, and B. E. Bouma, "Quantification of macrophage content in atherosclerotic plaques by optical coherence tomography," *Circulation*, vol. 107, pp. 113–119, Jan. 7, 2003.
- [3] G. J. Tearney, I. K. Jang, and B. E. Bouma, "Evidence of cholesterol crystals in atherosclerotic plaque by optical coherence tomographic (OCT) imaging," *Eur. Heart J.*, vol. 24, p. 1462, 2003.
- [4] G. J. Tearney, I. K. Jang, and B. E. Bouma, "Optical coherence tomography for imaging the vulnerable plaque," *J. Biomed. Opt.*, vol. 11, pp. 021002-1–021002-10, Mar./Apr. 2006.
- [5] I. K. Jang, B. E. Bouma, D. H. Kang, S. J. Park, S. W. Park, K. B. Seung, K. B. Choi, M. Shishkov, K. Schlendorf, E. Pomerantsev, S. L. Houser, H. T. Aretz, and G. J. Tearney, "Visualization of coronary atherosclerotic plaques in patients using optical coherence tomography: Comparison with intravascular ultrasound," *J. Amer. Coll. Cardiol.*, vol. 39, pp. 604–609, Feb. 20, 2002.
- [6] T. Kume, T. Akasaka, T. Kawamoto, Y. Ogasawara, N. Watanabe, E. Toyota, Y. Neishi, R. Sukmawan, Y. Sadahira, and K. Yoshida, "Assessment of coronary arterial thrombus by optical coherence tomography," *Amer. J. Cardiol.*, vol. 97, pp. 1713–1717, Jun. 15, 2006.
- [7] H. Yabushita, B. E. Bouma, S. L. Houser, H. T. Aretz, I. K. Jang, K. H. Schlendorf, C. R. Kauffman, M. Shishkov, D. H. Kang, E. F. Halpern, and G. J. Tearney, "Characterization of human atherosclerosis by optical coherence tomography," *Circulation*, vol. 106, pp. 1640–1645, Sep. 24, 2002.
- [8] J. Rieber, O. Meissner, G. Babaryka, S. Reim, M. Oswald, A. Koenig, T. M. Schiele, M. Shapiro, K. Theisen, M. F. Reiser, V. Klauss, and U. Hoffmann, "Diagnostic accuracy of optical coherence tomography and intravascular ultrasound for the detection and characterization of atherosclerotic plaque composition in ex vivo coronary specimens: A comparison with histology," *Coron. Artery Dis.*, vol. 17, pp. 425–430, Aug. 2006.
- [9] T. Kume, H. Okura, T. Kawamoto, T. Akasaka, E. Toyota, N. Watanabe, Y. Neishi, R. Sukmawan, Y. Sadahira, and K. Yoshida, "Relationship between coronary remodeling and plaque characterization in patients without clinical evidence of coronary artery disease," *Atherosclerosis*, vol. 197, pp. 799–805, 2008.
- [10] A. H. Association, *Heart Disease and Stroke Statistics – 2009 Update*. Dallas, TX: American Heart Association, 2009.
- [11] J. A. Schaar, J. E. Muller, E. Falk, R. Virmani, V. Fuster, P. W. Serruys, A. Colombo, C. Stefanadis, S. Ward Casscells, P. R. Moreno, A. Maseri, and A. F. van der Steen, "Terminology for

- high-risk and vulnerable coronary artery plaques. Report of a meeting on the vulnerable plaque, June 17 and 18, 2003, Santorini, Greece,” *Eur. Heart J.*, vol. 25, pp. 1077–1082, Jun. 2004.
- [12] F. D. Kolodgie, A. P. Burke, A. Farb, H. K. Gold, J. Yuan, J. Narula, A. V. Finn, and R. Virmani, “The thin-cap fibroatheroma: A type of vulnerable plaque: The major precursor lesion to acute coronary syndromes,” *Curr. Opin. Cardiol.*, vol. 16, pp. 285–292, Sep. 2001.
- [13] E. Falk, P. K. Shah, and V. Fuster, “Coronary plaque disruption,” *Circulation*, vol. 92, pp. 657–671, Aug. 1, 1995.
- [14] R. T. Lee and P. Libby, “The unstable atheroma,” *Arterioscler. Thromb. Vasc. Biol.*, vol. 17, pp. 1859–1867, Oct. 1997.
- [15] R. Virmani, F. D. Kolodgie, A. P. Burke, A. Farb, and S. M. Schwartz, “Lessons from sudden coronary death: A comprehensive morphological classification scheme for atherosclerotic lesions,” *Arterioscler. Thromb. Vasc. Biol.*, vol. 20, pp. 1262–1275, May 2000.
- [16] G. C. Cheng, H. M. Loree, R. D. Kamm, M. C. Fishbein, and R. T. Lee, “Distribution of circumferential stress in ruptured and stable atherosclerotic lesions. A structural analysis with histopathological correlation,” *Circulation*, vol. 87, pp. 1179–1187, Apr. 1993.
- [17] R. T. Lee, A. J. Grodzinsky, E. H. Frank, R. D. Kamm, and F. J. Schoen, “Structure-dependent dynamic mechanical behavior of fibrous caps from human atherosclerotic plaques,” *Circulation*, vol. 83, pp. 1764–1770, May 1991.
- [18] P. R. Moreno, V. H. Bernardi, J. Lopez-Cuellar, A. M. Murcia, I. F. Palacios, H. K. Gold, R. Mehran, S. K. Sharma, Y. Nemerson, V. Fuster, and J. T. Fallon, “Macrophages, smooth muscle cells, and tissue factor in unstable angina. Implications for cell-mediated thrombogenicity in acute coronary syndromes,” *Circulation*, vol. 94, pp. 3090–3097, Dec. 15, 1996.
- [19] A. Farb, A. P. Burke, A. L. Tang, T. Y. Liang, P. Mannan, J. Smialek, and R. Virmani, “Coronary plaque erosion without rupture into a lipid core. A frequent cause of coronary thrombosis in sudden coronary death,” *Circulation*, vol. 93, pp. 1354–1363, Apr. 1, 1996.
- [20] A. C. van derWal, A. E. Becker, C.M. van der Loos, and P. K. Das, “Site of intimal rupture or erosion of thrombosed coronary atherosclerotic plaques is characterized by an inflammatory process irrespective of the dominant plaque morphology,” *Circulation*, vol. 89, pp. 36–44, Jan. 1994.
- [21] P. Meier, R. Zbinden, M. Togni, P. Wenaweser, S. Windecker, B. Meier, and C. Seiler, “Coronary collateral function long after drug-eluting stent implantation,” *J. Amer. Coll. Cardiol.*, vol. 49, pp. 15–20, Jan. 2, 2007.
- [22] J. R. Nebeker, R. Virmani, C. L. Bennett, J. M. Hoffman, M. H. Samore, J. Alvarez, C. J. Davidson, J. M. McKoy, D.W. Raisch, B. K. Whisenant, P. R. Yarnold, S. M. Belknap, D. P. West, J. E. Gage, R. E. Morse, G. Gligoric, L. Davidson, and M. D. Feldman, “Hypersensitivity cases associated with drug-eluting coronary stents: A review of available cases from the Research on Adverse Drug Events and Reports (RADAR) project,” *J. Amer. Coll. Cardiol.*, vol. 47, pp. 175–181, Jan. 3, 2006.
- [23] M. Togni, S. Windecker, R. Cocchia, P. Wenaweser, S. Cook, M. Billinger, B. Meier, and O. M. Hess, “Sirolimus-eluting stents associated with paradoxical coronary vasoconstriction,” *J. Amer. Coll. Cardiol.*, vol. 46, pp. 231–236, Jul. 19, 2005.
- [24] A. K. Hassan, S. C. Bergheanu, T. Stijnen, B. L. van der Hoeven, J. D., Snoep, J.W. Plevier, M. J. Schalij, and J.W. Jukema. (2009, Jan. 21). Late stent malapposition risk is higher after drug-eluting stent compared with bare-metal stent implantation and associates with late stent thrombosis. *Eur. Heart J.* [Online].
- [25] B. D. MacNeill, H. C. Lowe, M. Takano, V. Fuster, and I. K. Jang, “Intravascular modalities for detection of vulnerable plaque: Current status,” *Arterioscler. Thromb. Vasc. Biol.*, vol. 23, pp. 1333–1342, Aug. 1, 2003.
- [26] A. J. Martin, L. K. Ryan, A. I. Gotlieb, R. M. Henkelman, and F. S. Foster, “Arterial imaging: Comparison of high-resolution US and MR imaging with histologic correlation,” *Radiographics*, vol. 17, pp. 189–202, Jan./Feb. 1997.
- [27] F. Prati, E. Arbustini, A. Labellarte, B. Dal Bello, L. Sommariva, M. T. Mallus, A. Pagano, and A. Boccanelli,

- “Correlation between high frequency intravascular ultrasound and histomorphology in human coronary arteries,” *Heart*, vol. 85, pp. 567–570, May 2001.
- [28] P. Schoenhagen and S. Nissen, “Understanding coronary artery disease: Tomographic imaging with intravascular ultrasound,” *Heart*, vol. 88, pp. 91–96, Jul. 2002.
- [29] J. M. Tobis, J. Mallery, D. Mahon, K. Lehmann, P. Zalesky, J. Griffith, J. Gessert, M. Moriuchi, M. McRae, M. L. Dwyer, N. Greep, and W.L. Henry, “Intravascular ultrasound imaging of human coronary arteries in vivo. Analysis of tissue characterizations with comparison to in vitro histological specimens,” *Circulation*, vol. 83, pp. 913–926, Mar. 1991.
- [30] P. G. Yock and P. J. Fitzgerald, “Intravascular ultrasound: State of the art and future directions,” *Amer. J. Cardiol.*, vol. 81, pp. 27E–32E, Apr. 9, 1998.
- [31] L. C. Correia, E. Atalar, M. D. Kelemen, O. Ocali, G. M. Hutchins, J. L. Fleg, G. Gerstenblith, E. A. Zerhouni, and J. A. Lima, “Intravascular magnetic resonance imaging of aortic atherosclerotic plaque composition,” *Arterioscler. Thromb. Vasc. Biol.*, vol. 17, pp. 3626–3632, Dec. 1997.
- [32] A. J. Martin and R. M. Henkelman, “Intravascular MR imaging in a porcine animal model,” *Magn. Reson. Med.*, vol. 32, pp. 224–229, Aug. 1994.
- [33] W. J. Rogers, J. W. Prichard, Y. L. Hu, P. R. Olson, D. H. Benckart, C. M. Kramer, D. A. Vido, and N. Reichek, “Characterization of signal properties in atherosclerotic plaque components by intravascular MRI,” *Arterioscler. Thromb. Vasc. Biol.*, vol. 20, pp. 1824–1830, Jul. 2000.
- [34] M. E. Brezinski, G. J. Tearney, B. E. Bouma, S. A. Boppart, M. R. Hee, E. A. Swanson, J. F. Southern, and J. G. Fujimoto, “Imaging of coronary artery microstructure (in vitro) with optical coherence tomography,” *Amer. J. Cardiol.*, vol. 77, pp. 92–93, Jan. 1, 1996.
- [35] D. Huang, E. A. Swanson, C. P. Lin, J. S. Schuman, W. G. Stinson, W. Chang, M. R. Hee, T. Flotte, K. Gregory, C. A. Puliafito, and J. G. Fujimoto, “Optical coherence tomography,” *Science*, vol. 254, pp. 1178–1181, Nov. 22, 1991.
- [36] M. Asakura, Y. Ueda, O. Yamaguchi, T. Adachi, A. Hirayama, M. Hori, and K. Kodama, “Extensive development of vulnerable plaques as a pancoronary process in patients with myocardial infarction: An angioscopic study,” *J. Amer. Coll. Cardiol.*, vol. 37, pp. 1284–1288, Apr. 2001.
- [37] K. Kodama, A. Hirayama, and Y. Ueda, “Usefulness of coronary angiography for the evaluation of hyperlipidemia,” *Nippon Rinsho*, vol. 60, pp. 927–932, May 2002.
- [38] K. Mizuno and H. Nakamura, “Percutaneous coronary angiography: Present role and future direction,” *Ann. Med.*, vol. 25, pp. 1–2, Feb. 1993.
- [39] Y. Ueda, M. Asakura, O. Yamaguchi, A. Hirayama, M. Hori, and K. Kodama, “The healing process of infarct-related plaques. Insights from 18 months of serial angioscopic follow-up,” *J. Amer. Coll. Cardiol.*, vol. 38, pp. 1916–1922, Dec. 2001.
- [40] S. Waxman, “Characterization of the unstable lesion by angiography, angiography, and intravascular ultrasound,” *Cardiol. Clin.*, vol. 17, pp. 295–305, May 1999.
- [41] W. Casscells, B. Hathorn, M. David, T. Krabach, W. K. Vaughn, H. A. McAllister, G. Bearman, and J. T. Willerson, “Thermal detection of cellular infiltrates in living atherosclerotic plaques: Possible implications for plaque rupture and thrombosis,” *Lancet*, vol. 347, pp. 1447–1451, May 25, 1996.
- [42] C. Stefanadis, K. Toutouzas, E. Tsiamis, C. Stratos, M. Vavuranakis, I. Kallikazaros, D. Panagiotakos, and P. Toutouzas, “Increased local temperature in human coronary atherosclerotic plaques: An independent predictor of clinical outcome in patients undergoing a percutaneous coronary intervention,” *J. Amer. Coll. Cardiol.*, vol. 37, pp. 1277–1283, Apr. 2001.
- [43] P. R. Moreno, R. A. Lodder, K. R. Purushothaman, W. E. Charash, W. N. O’Connor, and J. E. Muller, “Detection of lipid pool, thin fibrous cap, and inflammatory cells in human aortic atherosclerotic plaques by near-infrared spectroscopy,” *Circulation*, vol. 105, pp. 923–927, Feb. 26, 2002.
- [44] A. Christov, R. M. Korol, E. Dai, L. Liu, H. Guan, M. A. Bernards, P. B. Cavers, D. Susko, and A. Lucas, “In vivo optical analysis of quantitative changes in collagen and elastin during arterial

- remodeling,” *Photochem. Photobiol.*, vol. 81, pp. 457–466, Mar./Apr. 2005.
- [45] L. Marcu, Q. Fang, J. A. Jo, T. Papaioannou, A. Dorafshar, T. Reil, J. H. Qiao, J. D. Baker, J. A. Freischlag, and M. C. Fishbein, “In vivo detection of macrophages in a rabbit atherosclerotic model by time-resolved laser-induced fluorescence spectroscopy,” *Atherosclerosis*, vol. 181, pp. 295–303, Aug. 2005.
- [46] H. P. Buschman, G. Deinum, J. T. Motz, M. Fitzmaurice, J. R. Kramer, A. van der Laarse, A. V. Brusckhe, and M. S. Feld, “Raman microspectroscopy of human coronary atherosclerosis: Biochemical assessment of cellular and extracellular morphologic structures in situ,” *Cardiovasc. Pathol.*, vol. 10, pp. 69–82, Mar./Apr. 2001.
- [47] T. J. Romer, J. F. Brennan, 3rd, M. Fitzmaurice, M. L. Feldstein, G. Deinum, J. L. Myles, J. R. Kramer, R. S. Lees, and M. S. Feld, “Histopathology of human coronary atherosclerosis by quantifying its chemical composition with Raman spectroscopy,” *Circulation*, vol. 97, pp. 878–885, Mar. 10, 1998.
- [48] M. E. Brezinski, G. J. Tearney, B. E. Bouma, J. A. Izatt, M. R. Hee, E. A. Swanson, J. F. Southern, and J. G. Fujimoto, “Optical coherence tomography for optical biopsy. Properties and demonstration of vascular pathology,” *Circulation*, vol. 93, pp. 1206–1213, Mar. 15, 1996.
- [49] B. E. Bouma, G. J. Tearney, H. Yabushita, M. Shishkov, C. R. Kauffman, D. DeJoseph Gauthier, B. D. MacNeill, S. L. Houser, H. T. Aretz, E. F. Halpern, and I. K. Jang, “Evaluation of intracoronary stenting by intravascular optical coherence tomography,” *Heart*, vol. 89, pp. 317–320, Mar. 2003.
- [50] G. J. Tearney, M. E. Brezinski, S. A. Boppart, B. E. Bouma, N. Weissman, J. F. Southern, E. A. Swanson, and J. G. Fujimoto, “Images in cardiovascular medicine. Catheter-based optical imaging of a human coronary artery,” *Circulation*, vol. 94, pp. 3013–3013, Dec. 1, 1996.
- [51] J. G. Fujimoto, S. A. Boppart, G. J. Tearney, B. E. Bouma, C. Pitris, and M. E. Brezinski, “High resolution in vivo intra-arterial imaging with optical coherence tomography,” *Heart*, vol. 82, pp. 128–133, Aug. 1999.
- [52] I. K. Jang, G. Tearney, and B. Bouma, “Visualization of tissue prolapse between coronary stent struts by optical coherence tomography: Comparison with intravascular ultrasound,” *Circulation*, vol. 104, pp. 2754–2759, Nov. 27, 2001.
- [53] E. Grube, U. Gerckens, L. Buellesfeld, and P. J. Fitzgerald, “Images in cardiovascular medicine. Intracoronary imaging with optical coherence tomography: A new high-resolution technology providing striking visualization in the coronary artery,” *Circulation*, vol. 106, pp. 2409–2410, Oct. 29, 2002.
- [54] H. M. Garcia-Garcia, N. Gonzalo, E. Regar, and P. W. Serruys, “Virtual histology and optical coherence tomography: From research to broad clinical application,” *Heart*, vol. 95, pp. 1362–1374, 2009.
- [55] N. Gonzalo, P. W. Serruys, T. Okamura, H. M. van Beusekom, H. M. Garcia-Garcia, G. van Soest, W. van der Giessen, and E. Regar, “Optical coherence tomography patterns of stent restenosis,” *Amer. Heart J.*, vol. 158, pp. 284–293, Aug. 2009.
- [56] G. J. Tearney, M. E. Brezinski, B. E. Bouma, S. A. Boppart, C. Pitris, J. F. Southern, and J. G. Fujimoto, “In vivo endoscopic optical biopsy with optical coherence tomography,” *Science*, vol. 276, pp. 2037–2039, 1997.
- [57] V. Fuster, “Lewis A. Conner Memorial Lecture. Mechanisms leading to myocardial infarction: Insights from studies of vascular biology,” *Circulation*, vol. 90, pp. 2126–2146, Oct. 1994.
- [58] C. L. Lendon, M. J. Davies, G. V. Born, and P. D. Richardson, “Atherosclerotic plaque caps are locally weakened when macrophage density is increased,” *Atherosclerosis*, vol. 87, pp. 87–90, Mar. 1991.
- [59] P. R. Moreno, E. Falk, I. F. Palacios, J. B. Newell, V. Fuster, and J. T. Fallon, “Macrophage infiltration in acute coronary syndromes. Implications for plaque rupture,” *Circulation*, vol. 90, pp. 775–778, Aug. 1994.
- [60] G. J. Tearney, I. K. Jang, D. H. Kang, H. T. Aretz, S. L. Houser, T. J. Brady, K. Schlendorf, M. Shishkov, and B. E. Bouma, “Porcine coronary imaging in vivo by optical coherence tomography,” *Acta Cardiol.*, vol. 55, pp. 233–237, Aug. 2000.

- [61] P. Barlis, P.W. Serruys, N. Gonzalo, W. J. van derGiessen, P. J. de Jaegere, and E. Regar, "Assessment of culprit and remote coronary narrowings using optical coherence tomography with long-term outcomes," *Amer. J. Cardiol.*, vol. 102, pp. 391–395, Aug. 15, 2008.
- [62] I. K. Jang, G. J. Tearney, B. MacNeill, M. Takano, F. Moselewski, N. Iftima, M. Shishkov, S. Houser, H. T. Aretz, E. F. Halpern, and B. E. Bouma, "In vivo characterization of coronary atherosclerotic plaque by use of optical coherence tomography," *Circulation*, vol. 111, pp. 1551–1555, Mar. 29, 2005.
- [63] S. Chia, O. C. Raffel, M. Takano, G. J. Tearney, B. E. Bouma, and I. K. Jang, "In vivo comparison of coronary plaque characteristics using optical coherence tomography in women vs. men with acute coronary syndrome," *Coron. Artery Dis.*, vol. 18, pp. 423–427, Sep. 2007.
- [64] T. Kubo, T. Imanishi, S. Takarada, A. Kuroi, S. Ueno, T. Yamano, T. Tanimoto, Y. Matsuo, T. Masho, H. Kitabata, K. Tsuda, Y. Tomobuchi, and T. Akasaka, "Assessment of culprit lesion morphology in acute myocardial infarction: Ability of optical coherence tomography compared with intravascular ultrasound and coronary angiography," *J. Amer. Coll. Cardiol.*, vol. 50, pp. 933–939, Sep. 4, 2007.
- [65] O. C. Raffel, G. J. Tearney, D. D. Gauthier, E. F. Halpern, B. E. Bouma, and I. K. Jang, "Relationship between a systemic inflammatory marker, plaque inflammation, and plaque characteristics determined by intravascular optical coherence tomography," *Arterioscler. Thromb. Vasc. Biol.*, vol. 27, pp. 1820–1827, Aug. 2007.
- [66] K. Toutouzas, S. Vaina, M. I. Riga, and C. Stefanadis, "Evaluation of dissection after coronary stent implantation by intravascular optical coherence tomography," *Clin. Cardiol.*, vol. 32, pp. E47–E48, 2009.
- [67] E. Regar, J. Schaar, and P. W. Serruys, "Images in cardiology. Acute recoil in sirolimus eluting stent: Real time, in vivo assessment with optical coherence tomography," *Heart*, vol. 92, p. 123, Jan. 2006.
- [68] O. C. Raffel, J. C. Hannan, and I. K. Jang, "Coronary stent malapposition as a result of a post-stenotic aneurysm detected by optical coherence tomography," *J. Invasive Cardiol.*, vol. 18, pp. 561–562, Nov. 2006.
- [69] T. Sawada, J. Shite, T. Shinke, S. Watanabe, H. Otake, D. Matsumoto, Y. Imuro, D. Ogasawara, O. L. Paredes, and M. Yokoyama, "Persistent malapposition after implantation of sirolimus-eluting stent into intramural coronary hematoma: Optical coherence tomography observations," *Circ. J.*, vol. 70, pp. 1515–1519, Nov. 2006.
- [70] M. Takano, I. K. Jang, and K. Mizuno, "Neointimal proliferation around malapposed struts of a sirolimus-eluting stent: Optical coherence tomography findings," *Eur. Heart J.*, vol. 27, pp. 1763–1763, Aug. 2006.
- [71] E. Regar, H. M. van Beusekom, W. J. van der Giessen, and P.W. Serruys, "Images in cardiovascular medicine. Optical coherence tomography findings at 5-year follow-up after coronary stent implantation," *Circulation*, vol. 112, pp. e345–e346, Dec. 6, 2005.
- [72] P. Barlis, J. Tanigawa, and C. Di Mario, "Coronary bioabsorbable magnesium stent: 15-month intravascular ultrasound and optical coherence tomography findings," *Eur. Heart J.*, vol. 28, p. 2319, May 7, 2007.
- [73] R. Gupta, O. C. Raffel, and I. K. Jang, "Severe intimal hyperplasia after sirolimus eluting stent deployment: Evaluation by optical coherence tomography," *Heart*, vol. 93, p. 754, Jun. 2007.
- [74] J. Tanigawa, P. Barlis, and C. Di Mario, "Do unapposed stent struts endothelialise? In vivo demonstration with optical coherence tomography," *Heart*, vol. 93, pp. 378–378, Mar. 2007.
- [75] M. Takano, S. Inami, I. K. Jang, M. Yamamoto, D. Murakami, K. Seimiya, T. Ohba, and K. Mizuno, "Evaluation by optical coherence tomography of neointimal coverage of sirolimus-eluting stent three months after implantation," *Amer. J. Cardiol.*, vol. 99, pp. 1033–1038, Apr. 15, 2007.
- [76] E. Camenzind, P. G. Steg, and W. Wijns, "Stent thrombosis late after implantation of first-generation drug-eluting stents: A cause for concern," *Circulation*, vol. 115, pp. 1440–1455, Mar. 20, 2007.
- [77] S. H. Yun, G. J. Tearney, B. J. Vakoc, M. Shishkov, W. Y. Oh, A. E. Desjardins, M. J. Suter, R. C. Chan, J. A. Evans, I. K. Jang, N. S. Nishioka, J.

- F. de Boer, and B. E. Bouma, Comprehensive volumetric optical microscopy in vivo,” *Nat. Med.*, vol. 12, pp. 1429–1433, 2006.
- [78] S. H. Yun, G. J. Tearney, J. F. de Boer, N. Ifima, and B. E. Bouma, “High-speed optical frequency-domain imaging,” *Opt. Exp.*, vol. 11, pp. 2953–2963, 2003.
- [79] M. Choma, M. Sarunic, C. Yang, and J. Izatt, “Sensitivity advantage of swept source and Fourier-domain optical coherence tomography,” *Opt. Exp.*, vol. 11, pp. 2183–2189, Sep. 8, 2003.
- [80] R. Huber, D. C. Adler, and J. G. Fujimoto, “Buffered Fourier-domain mode locking: Unidirectional swept laser sources for optical coherence tomography imaging at 370000 lines/s,” *Opt. Lett.*, vol. 31, pp. 2975–2977, Oct. 15, 2006.
- [81] R. Huber, M. Wojtkowski, and J. G. Fujimoto, “Fourier-Domain Mode Locking (FDML): A new laser operating regime and applications for optical coherence tomography,” *Opt. Exp.*, vol. 14, pp. 3225–3237, Apr. 17, 2006.
- [82] Y. Mao, S. Sherif, C. Flueraru, and S. Chang, “3×3 Mach-Zehnder interferometer with unbalanced differential detection for full-range sweptsource optical coherence tomography,” *Appl. Opt.*, vol. 47, pp. 2004–2010, Apr. 20, 2008.
- [83] M. Sarunic, M. A. Choma, C. Yang, and J. A. Izatt, “Instantaneous complex conjugate resolved spectral domain and swept-source OCT using 3 × 3 fiber couplers,” *Opt. Exp.*, vol. 13, pp. 957–967, Feb. 7, 2005.
- [84] M. V. Sarunic, B. E. Applegate, and J. A. Izatt, “Real-time quadrature projection complex conjugate resolved Fourier-domain optical coherence tomography,” *Opt. Lett.*, vol. 31, pp. 2426–2428, Aug. 15, 2006.
- [85] B. J. Vakoc, S. H. Yun, G. J. Tearney, and B. E. Bouma, “Elimination of depth degeneracy in optical frequency-domain imaging through polarization-based optical demodulation,” *Opt. Lett.*, vol. 31, pp. 362–364, Feb. 1, 2006.
- [86] S. Yun, G. Tearney, J. de Boer, and B. Bouma, “Removing the depth degeneracy in optical frequency-domain imaging with frequency shifting,” *Opt. Exp.*, vol. 12, pp. 4822–4828, Oct. 4, 2004.
- [87] G. J. Tearney, S. Waxman, M. Shishkov, B. J. Vakoc, M. J. Suter, M. I. Freilich, A. E. Desjardins, W. Y. Oh, L. A. Bartlett, M. Rosenberg, and B. E. Bouma, “3-D coronary artery microscopy by intracoronary optical frequency-domain imaging,” *JACC Cardiovasc. Imag.*, vol. 1, pp. 752–761, Nov. 2008.
- [88] J. F. de Boer, T. E. Milner, M. J. C. van Gemert, and J. S. Nelson, “2-D birefringence imaging in biological tissue by polarization-sensitive optical coherence tomography,” *Opt. Lett.*, vol. 22, pp. 934–936, 1997.
- [89] M. J. Everett, K. Schoenenberger, B. W. Colston, and L. B. da Silva, “Birefringence characterization of biological tissue by use of optical coherence tomography,” *Opt. Lett.*, vol. 23, pp. 228–230, 1998.
- [90] J. F. de Boer and T. E. Milner, “Review of polarization sensitive optical coherence tomography and Stokes vector determination,” *J. Biomed. Opt.*, vol. 7, pp. 359–371, Jul. 2002.
- [91] S. K. Nadkarni, M. C. Pierce, B. H. Park, J. F. de Boer, P. Whittaker, B. E. Bouma, J. E. Bressner, E. Halpern, S. L. Houser, and G. J. Tearney, “Measurement of collagen and smooth muscle cell content in atherosclerotic plaques using polarization-sensitive optical coherence tomography,” *J. Amer. Coll. Cardiol.*, vol. 49, pp. 1474–1481, Apr. 3, 2007.
- [92] B. Park, M. C. Pierce, B. Cense, and J. de Boer, “Real-time multifunctional optical coherence tomography,” *Opt. Exp.*, vol. 11, pp. 782–793, Apr. 7, 2003.
- [93] B. H. Park, M. C. Pierce, B. Cense, and J. F. de Boer, “Jones matrix analysis for a polarization-sensitive optical coherence tomography system using fiber-optic components,” *Opt. Lett.*, vol. 29, pp. 2512–2514, Nov. 1, 2004.
- [94] B. H. Park, M. C. Pierce, B. Cense, S. H. Yun, M. Mujat, G. J. Tearney, B. E. Bouma, and J. F. de Boer, “Real-time fiber-based multifunctional spectral-domain optical coherence tomography at 1.3 μm,” *Opt. Exp.*, vol. 13, pp. 3931–3944, 2005.
- [95] C. E. Saxer, J. F. de Boer, B. H. Park, Y. Zhao, Z. Chen, and J. S. Nelson, “High-speed fiber-based polarization-sensitive optical coherence tomography of in vivo human skin,” *Opt. Lett.*, vol. 25, pp. 1355–1357, 2000.
- [96] B. H. Park, C. Saxer, S. M. Srinivas, J. S. Nelson, and J. F. de Boer, “In vivo burn depth determination by high-speed fiber-based polarization sensitive optical coherence tomography,” *J. Biomed. Opt.*, vol. 6, pp. 474–479, Oct. 2001.

- [97] J. Zhang, W. Jung, J. S. Nelson, and Z. Chen, "Full range polarization sensitive Fourier-domain optical coherence tomography," *Opt. Exp.*, vol. 12, pp. 6033–6039, 2004.
- [98] W. Y. Oh, S. H. Yun, B. J. Vakoc, M. Shishkov, A. E. Desjardins, B. H. Park, J. F. de Boer, G. J. Tearney, and B. E. Bouma, "High-speed polarization sensitive optical frequency-domain imaging with frequency multiplexing," *Opt. Exp.*, vol. 16, pp. 1096–1103, 2008.
- [99] J. Su, J. Zhang, L. Yu, H. G. Colt, M. Brenner, and Z. Chen, "Realtime swept source optical coherence tomography imaging of the human airway using a microelectromechanical system endoscope and digital signal processor," *J. Biomed. Opt.*, vol. 13, pp. 030506-1–030506-3, May/Jun. 2008.
- [100] A. Desjardins, B. Vakoc, M. Suter, G. Tearney, and B. Bouma, "Real-time FPGA processing for high-speed optical frequency-domain imaging," *IEEE Trans. Med. Imag.*, vol. 28, no. 9, pp. 1468–1472, Sep. 2009.
- [101] V. Westphal, S. Yazdanfar, A. M. Rollins, and J. A. Izatt, "Real-time, high velocity-resolution color Doppler optical coherence tomography," *Opt. Lett.*, vol. 27, pp. 34–36, Sep. 2009.
- [102] S. Yan, D. Piao, Y. Chen, and Q. Zhu, "Digital signal processor-based real-time optical Doppler tomography system," *J. Biomed. Opt.*, vol. 9, pp. 454–463, May/Jun. 2004.
- [103] Y. Yasuno, S. Makita, T. Endo, G. Aoki, H. Sumimura, M. Itoh, and T. Yatagai, "One-shot-phase-shifting Fourier-domain optical coherence tomography by reference wavefront tilting," *Opt. Exp.*, vol. 12, pp. 6184–6191, Dec. 13, 2004.
- [104] G. T. Bonnema, K. O. Cardinal, S. K. Williams, and J. K. Barton, "An automatic algorithm for detecting stent endothelialization from volumetric optical coherence tomography datasets," *Phys. Med. Biol.*, vol. 53, pp. 3083–3098, Jun. 21, 2008.
- [105] A. Wahle, J. J. Lopez, M. E. Olszewski, S. C. Vigmostad, K. B. Chandran, J. D. Rossen, and M. Sonka, "Plaque development, vessel curvature, and wall shear stress in coronary arteries assessed by X-ray angiography and intravascular ultrasound," *Med. Image Anal.*, vol. 10, pp. 615–631, Aug. 2006.
- [106] A. Wahle and M. Sonka, "Coronary plaque analysis by multimodality fusion," *Stud. Health Technol. Inf.*, vol. 113, pp. 321–359, 2005.