

Venous blood clot structure characterization using scattering operator

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Abstract—Deep venous thrombosis (DVT) occurs when a blood clot appear within a deep vein, usually in the legs. The main complication is pulmonary embolism (PE) which is the third cause of vascular death after myocardial infarction and cardiovascular event. DVT onset is multifactorial (immobilization, surgery, age, cancers, genetic variations) and it is mostly diagnosed via ultrasound. In our project, we are interested in a new approach, which consists in using ultrasound and elastography to assess the mechanical properties of blood clots and to identify the thrombosis triggering factors. This means characterizing its structure, establishing his age, the cause of its formation and the risk of PE. In this paper, we aim to analyze the clot texture using the scattering operator [1] which combines wavelet transform convolutions with non-linear modulus and averaging operators. The scattering operator is showing promising results in signal processing and especially in image classification [2]. Therefore, we will apply this operator to our database and discuss the results of the simulation at the end of this manuscript.

Keywords—deep venous thrombosis (DVT); clot; ultrasound imaging; elastography; scattering operator; wavelet

I. INTRODUCTION

The abnormal formation of a blood clot in a blood vessel is named thrombosis. Symptoms related to thrombosis depend on location, size and structure. Our project considers only Deep Venous Thrombosis (DVT) *i.e.* blood clots that block partially or totally deep veins of the legs (popliteal, femoral, and iliac). The main complication occurs when a clot fragment comes off and travels to the lung. This process is named pulmonary embolism (PE). Virchow's triad [3] describes three physiopathological mechanisms that contribute, isolated or combined, to the development of DVT: (a) stasis (e.g. immobilization), (b) endothelial injury (e.g. catheter) and (c) hypercoagulability (e.g. hormone, cancer, genetic variations). More precisions on blood clot formation and risk factors are provided in Section II.

The three main signs of DVT are calf or ankle swelling, leg warm to the touch and leg pain or tenderness. These signs are not specific to DVT and some people suffering from DVT may have none of these symptoms. In Section III, different diagnose techniques used by doctors are presented (e.g. angiography, ultrasound). The detection of this pathology is relatively simple yet; it is a lot more difficult to identify the thrombosis origins,

age and to estimate the risk of PE. In the literature, we can find studies linking thrombosis maturity to clot elasticity ([4], [5], and [6]), determining the impact of genetic variations on the onset of a DVT ([7] and [8]) or estimating treatment efficiency ([9] and [10]). The objective of our project is to analyze the blood clot structure with the help of ultrasound and elastometry techniques in order to estimate the age, the origins and the risk of PE. Ultimately, a major stake will be the detection of cancer in an early stage on a patient victim of DVT. Section IV describes the scattering operator [1] and its simulation results obtain on clot ultrasound images. This multiscale operator is based on wavelet filter banks and modulus rectifiers. The originality of this manuscript is to evaluate this technique on our database. Indeed, the literature [2] and our previous industrial project show that the scattering operator have really good performances on texture classification.

II. BLOOD CLOT FORMATION AND BREAKDOWN

A. Blood circulation [11]

Blood flows one-way through a closed system formed by different veins and arteries. Oxygenated blood flows from the heart to various organs through arteries which have thick walls. Veins have thinner and elastic walls and take over from arteries to pull up oxygen impoverished blood back to the heart thank to four: (a) the heartbeat which maintains a continuous flow, (b) the diaphragmatic or deep breathing, (c) the muscle pump system (calf muscular contraction) and (d) the venous pump of the foot which is the first step in venous return of blood when walking. Moreover, veins include a valve system to avoid blood reflux. Prolonged immobilization due to plaster cast, bed rest or long distance flights slows down blood circulation and encourages the growth of venous thrombosis.

B. Hemostasis and fibrinolysis [12]

The physiological process keeping blood within veins and stopping the bleeding in case of vessel injuries is called hemostasis. Hemostasis consists of three main phases: (a) vascular spasm, (b) primary and (c) secondary hemostasis. The first one corresponds to the reduction of damaged vessel diameter so that the bleeding progressively slows down. Next, platelets begin to adhere to the cut edges of the vessel and release chemicals to attract even more platelets. The platelet plug formation stops the external bleeding and this second phase is called primary hemostasis. The secondary hemostasis defines the following mechanism: small molecules, called

clotting factors, begin to create the clot and form a collagen fiber called fibrin. When the vessel is being repaired, a process named fibrinolysis starts dissolving the clot and prevents it from growing and causing thrombosis. Hemostasis or fibrinolysis disorder is therefore one of the risk factors for venous thrombosis.

C. Thrombosis and symptoms [11], [12]

Thrombosis occurs when a blood clot is formed and bleeding stop process does not start or when the fibrinolysis step is deficient. It manifests as an inflammation of a stamped or obstructed vessel due to the clot. Venous thrombosis mostly appears in the legs. There are two levels of thrombosis: superficial and deep thrombosis. The first one comes out in the superficial veins *i.e.* in the veins near the skin and usually causes no fever, no infection and no swelling. It is dangerous because it can hide a deeper thrombosis or evolve to reach the deep venous network.

Deep Venous Thrombosis (DVT) is extremely dangerous because a piece or the entire deep vein clot could break off, be carried by the blood to the lung and cause a pulmonary embolism (PE). The clot formation often begins in the calf veins, where the blood flow may slow down (valves, collateral veins) and extends to the knee or thigh veins. The risk of PE is that the clot gets closer to the heart. When a deep vein is obstructed by a clot, the blood cannot flow back to the heart through this vein, this creates a hypertension upstream part of the clot, so the blood has no other choice than to flow through the superficial venous network.

DVT can be revealed by three main signs: swelling of the calf or the ankle, leg warm to the touch and leg pain or tenderness. The symptoms of a DVT depend on the inflammatory response extend around the clot and on its size. The patient could also have numbing, cramps, a sensation of heavy legs, pain on palpation and/or bluish skin discoloration. At a confirmed stage, DVT may go along with fever, edema, ulcer, tachycardia or even a complete functional impotence. Sometimes there is no symptom, making this disease even more dangerous. Moreover, relapse is frequent and the patient can have post-thrombotic symptoms *i.e.* long-term complications.

D. Risk factors

In normal conditions, there is a balance in the blood circulation among molecules of the coagulant and anticoagulant systems. Many physiopathological mechanisms can unbalance the blood circulation and create a venous thrombosis. Virchow [3] has given a substantial contribution to our knowledge about the venous thromboembolism. Virchow's triad describes three mechanisms that contribute, isolated or combined, to the development of DVT: (a) stasis, (b) endothelial injury and (c) hypercoagulability

The first term of the Virchow's triad is stasis and gathers all factors responsible for the blood slow down. Extended immobilization due to a disease, a bed rest or a labor, plaster cast, heart failure, varicose vein and venous constriction because of a prolonged sitting, a long distance flight or a cancer contribute to reduce the blood flow and stimulate the appearance of thrombosis. For instance, statistics on venous thrombosis showed an incidence of two to four per ten thousand passengers of a flight over five hours [13]. Smoking also increases the risk as it stimulates the production of fibrinogen in the blood (and hence platelet aggregation and

coagulation) and makes the blood more viscous. As a reaction, the number of red and white blood cells goes up in order to make up for oxygen insufficiency.

The second group of factors includes surgery, catheter, traumatism and age. These factors degrade venous walls; therefore an infection can cause an inflammation, so the return of blood in the vein can produce a thrombosis. Risks of DVT are stronger after a hospital discharge and with elderly patients. Statistics [13] show that the risks, without preventive thrombosis in medicine, increase in the range of ten to twenty percent in the four to five weeks following a hospital discharge. In the same way, risks become twice larger every ten years after the age of 40.

Hypercoagulability constitutes the third part of the triad and incorporates all mechanisms reinforcing coagulation or disturbing anticoagulation. It can be acquired factors (such as pregnancy, hormone therapy, cancer), inflammatory disorders or inherited factors (Factor V Leiden mutation, protein C or S deficiency, antithrombin deficiency). Pregnant women or people with hormonal treatment have two to five times more likely to develop a DVT [13].

E. Diagnosis and treatment [12]

In order to reduce the risk of PE, it is vital to diagnose as soon as possible the DVT. However, this task seems to be difficult because the signs and symptoms associated with the DTV are not specific to this disease. D-dimer is a fibrin degradation that occurs in the blood after a clot is degraded by fibrinolysis. While the measure of D-dimer concentration allows excluding thromboembolic disease where the probability is low, a positive result does not always indicate thrombosis. Test using ultrasound dispel doubt of DVP: Doctors can view the blood network, see the blood flow and check on the veins' compressibility. A vein with a blood clot is relatively incompressible and is more echogenic than a free vein.

III. DIAGNOSTICAL TECHNIQUES

The difficulties in the diagnosis of the DVT are related to its characteristics often asymptomatic and the lack of specific clinical signs. The following items give the most commonly used methods to detect a DVP and to analyze the blood clot:

A. Common techniques

- By injected a contrast medium, venography (or phlebography or venous angiography) uses X-rays to examine the veins. Nowadays, this procedure is rarely applied because of its cost and its invasiveness. However, it still remains the gold standard for diagnosing DVT with imaging means.
- Using Magnetic Resonance Imaging (MRI), Magnetic Resonance Angiography (MRA) visualizes the blood vessels and examines the abnormalities of the arteries and less commonly of the veins. This method mostly involves intravenous contrast agents.
- Computed Tomography Angiography (CTA) is also an invasive technique but displays the anatomical detail of blood vessels more precisely than MRA or ultrasound. Its main application is screening for arterial disease because it is safer and less time-consuming than angiography.

- The main imaging technique to explore the deep venous network is Doppler ultrasonography. Ultrasonography allows to view the veins and to test the presence of a clot (compressibility and echogenic mark). The Doppler extension puts the blood flow up (or the absence of flow if the vein is blocked). The frequency of the used sound waves varies between 50 Hz and 20 kHz depending on the expected exploring depth [14].

B. A new approach : elastography/metry

In the medical context, the main application is the diagnosis of hepatic fibrosis because the liver gets harder when the fibrosis gets more severe. In old days, palpation was used to estimate the hardness. Recently, several systems can precisely measure and create a map of the organ hardness (elastography).

1) Principle

Elastometry consists in estimating the hardness, or the elasticity, of human tissues, e.g. their resistance when a mechanical force is applied on it: the harder a tissue is, the more elastic it is. A static external stress σ (in pascal Pa), applied to the surface of a solid, is linearly proportional to its fractional extension ε (non-dimensional) by the modulus of elasticity E (in Pa). This principle is named Hooke's law [4]:

$$\sigma = \varepsilon E \quad (1)$$

Human soft tissues can distort under the influence of two types of mechanical waves: compressional and shear waves. The first type is also called longitudinal waves because the particle displacement is parallel to the direction of wave propagation. The second type is equivalently called transverse waves because the particle displacement is perpendicular to the direction of the wave propagation. The velocity of these waves is directly connected to the elastic modulus E (or Young's modulus). In soft biological tissues [15], the compressional velocity is far higher (≈ 1500 m/s) than the shear wave velocity (≈ 10 m/s), so the Young's modulus can be approximated using the following equation:

$$E \approx 3\rho c_s^2 \quad (2)$$

where ρ is the volume density (kg/m^3) and c_s the shear wave velocity (m/s). The volume density is assumed to be constant (1000 kg/m^3 which is the water density) even if it is actually different from one tissue to another (fat $\approx 950 \text{ kg/m}^3$, blood $\approx 1025 \text{ kg/m}^3$, liver $\approx 1060 \text{ kg/m}^3$, muscle $\approx 1070 \text{ kg/m}^3$ and bone between 1380 kg/m^3 and 1810 kg/m^3 [16]).

2) Current systems

Elastometry systems do not measure directly the hardness of the human tissue but estimate the velocity of the shear waves. These systems send either a mechanical or an acoustic impulse to generate the shear waves and follow their propagation using ultrasound, *i.e.* compressional waves. Ultrasonic echoes are analyzed in order to determine the velocity of the shear waves, and hence the elasticity. Currently, there are several systems able to quantify the hardness of biological tissues:

- Fibroscan (Echosens): this system sends a mechanical impulse to create the shear waves and is mainly used to diagnose fibrosis and especially hepatic fibrosis. This method is quantitative.
- Virtual Touch Imaging (Siemens): the impulse is, here, ultrasonic. Ultrasound imaging allows the user to

define the region of interest (ROI) on the target and the system computes the velocity of the shear waves in the ROI.

- Aixplorer (Supersonic Image): it operates similarly to the Virtual Touch Imaging system (ultrasonic shear waves and ROI) but the user can also display an elasticity map in a predefined window and get the mean elasticity in a ROI within this window. This technics is therefore both quantitative and qualitative.
- Aplio 500 (Toshiba): this system is close to the Aixplorer system and is used to create the database and the exploration in this study.

IV. SIMULATION

By applying the scattering operator, we try to analyze the clot structure using ultrasound images. The database combines ultrasound images from 18 patients suffering from thrombosis. For four of them, we can follow the evolution of the DTV taking three months later. We choose to apply the scattering operator [1] on our database because this algorithm has given very good results on our previous industrial project. The goal of this project is to classify acoustic images of the seabed.

A. Scattering operator [1]

The challenge of automatically classify signals and especially images resides in the high variability within a same class of signals. This variability is often uninformative in the sense that it does not characterize a class change. The scattering operator aims at reducing this variability by creating a "translation invariant image representation, which is stable to deformations and preserves high frequency information for classification" [1]. A scattering transform computes coefficients by iterating wavelet transforms and modulus operator (see Eq (4)).

The translation invariance is obtained using a low-pass filter:

$$\phi_J(u) = 2^{-2J}\phi(2^{-J}u) \quad (3)$$

where 2^J is the maximum scale and u stands for the spatial position vector and ϕ is a scaling function named father wavelet. The first coefficient of the scattering transform of an image x is defined by the following equation:

$$S[\emptyset]x = x * \phi_J \quad (4)$$

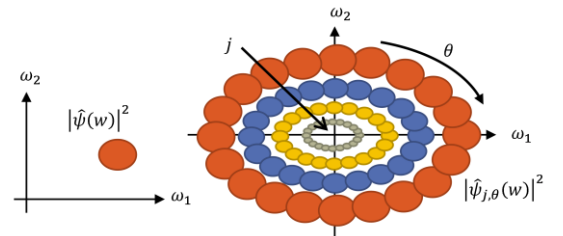


Figure 1. Frequency support of the mother wavelet on the left side and of the child wavelets on the right side ($\hat{\psi}_\lambda$ is the Fourier transform of ψ_λ)

The high-frequency information affected by this filter is recovered via two-dimensional wavelets. These wavelets are obtained by scaling and rotating a band-pass filter ψ (see Figure 1). The number of rotations and scales are key parameters of the scattering transform. During the simulation, they are respectively fixed at four rotations and three scales (based on the literature). These located wavelets are defined,

for each scale $0 < j < J$ and an orientation θ , by the following equation:

$$\psi_\lambda(u) = 2^{-2j}\psi(2^{-j}r_\theta^{-1}u) \quad (5)$$

where λ can be assimilated at a frequency variable: $\lambda = 2^{-j}r_\theta$

and r_θ is the rotation matrix $r_\theta = \begin{bmatrix} \cos(\theta) & -\sin(\theta) \\ \sin(\theta) & \cos(\theta) \end{bmatrix}$.

The wavelet transform is stable to small deformation and invertible if the rotated and scaled wavelet filters cover the whole frequency plane. In the simulation, the Morlet wavelet family is used. The use of the norm $L_1(\mathbb{R}^2)$ on the wavelet coefficient modulus makes the representation translation - invariant :

$$\|x * \psi_\lambda\|_1 = \int_{\mathbb{R}} |x * \psi_\lambda(u)| du \quad (6)$$

The first layer of the scattering transform is thus defined by applying the average filter ϕ_j :

$$S[\lambda_1]x = |x * \psi_{\lambda_1}| * \phi_j \quad (7)$$

The integration, which removes all non-zero frequency components, causes an information loss. However, this information can be recovered by calculating the wavelet coefficients of $|x * \psi_{\lambda_1}|$. Therefore their $L_1(\mathbb{R}^2)$ norms define a larger family of invariants (second layer):

$$S[\lambda_1, \lambda_2]x = \left| |x * \psi_{\lambda_1}| * \psi_{\lambda_2} \right| * \phi_j \quad (7)$$

Further iteration on the wavelet transforms and the modulus operators enables evaluating more translation invariant coefficients:

$$S[p]x = \left| \left| |x * \psi_{\lambda_1}(u)| * \psi_{\lambda_2}(u) \right| \dots * \psi_{\lambda_m}(u) \right| * \phi_j(u) \quad (8)$$

where $p = (\lambda_1, \lambda_2, \dots, \lambda_m)$ is the set of all paths and m the number of layers.

In practice, only the first and the second layers are computed. These coefficients can be displayed as piecewise constant functions equal to $S[p]x$ over each frequency subset. Figure 2 represents the scattering transform of an image with a striped pattern. The maximum coefficient corresponds to the orientation and the frequency (or scale) of the stripes: here the maximums correspond on the orientation 45° and the smallest scale.

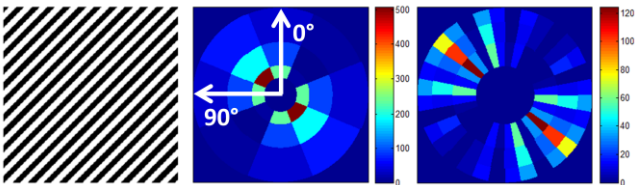


Figure 2. Scattering representation of stripes in the frequency plane: on the first layer (middle), each rotated quadrant has an area proportional to $2^{j'}$; on the second layer (right) each quadrant of the first is subdivided into a partition of subsets proportional to 2^{j^2} .

B. Application on the entire ultrasound images

Firstly, the scattering operator is applied on the entire image/ without specific preprocessing. The first row of Figures 3 presents three ultrasound images, two of which come from the same patient. The blood clot is encircled by an ellipse drawn by a medical expert. The second and the third row of Figures 3 show the associated scattering coefficients displayed in the frequency plane at the first and second order. The

coefficients of the two images taken from the same patient are very similar. In addition, we can see that the second patient's coefficients are quite different from those of the first patient. To distinguish images from different patients seems possible using the scattering operator. Indeed, the size of the blood clot and the contrast between the vessels and the rest of the image differ among the patients.

However, the images do not only content the obstructed vein but also the artery and human tissue. Henceforth, the scattering operator does not allow us to characterize the blood clot. In the next section, the scattering operator is applied only on blood clot images.

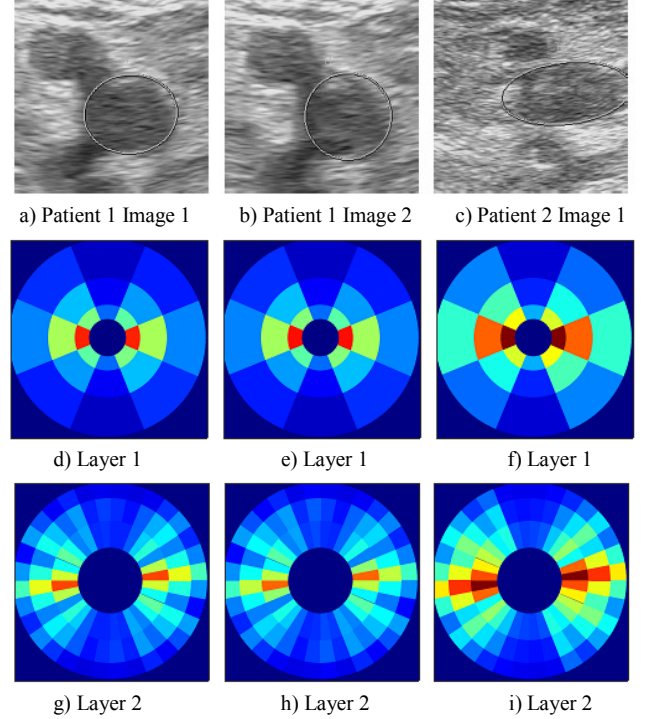


Figure 3. Three ultrasound images taken from two different patients (the blood clots are encircled); the first and the second layers of the scattering coefficients are represented in the frequency plane.

C. Application on the ultrasound image of the clot

1) Extraction of the clot on the image

As depicted on Figure 3, the blood clot is encircled by an ellipse made by a medical expert. To only analyze the clot structure, the largest square contained in the ellipse is determined. To do this, firstly, the ellipse is segmented using its specific pixel color. Then, our method closes the edge of the ellipse if necessary and fills the ellipse. We estimate next the ellipse parameters (centroid O , orientation α , major a and minor b axes) and compute the coordinates of the square corners using the following system:

$$\frac{X^2}{a^2} + \frac{Y^2}{b^2} = 1 \quad (9)$$

$$\text{where} \quad X = c(\cos\alpha - \sin\alpha)$$

$$\text{and} \quad Y = c(\cos\alpha + \sin\alpha)$$

The width of the largest square in the ellipse corresponds to the variable c . The Figure 4 illustrates our idea and the different variables.

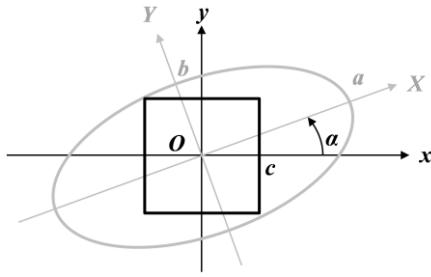


Figure 4. Extraction of the largest square inside an oriented ellipse.

2) Application of the scattering operator

Once the smaller images centered on the blood clots have been extracted from the original ultrasound images, the scattering operator is applied on them. The results are difficult to be interpreted because the scattering coefficients seem to be quite similar for different patients. In some cases, we observed that the scattering coefficients from two different patients (Figure 5 g) and i)) are more similar than those from the same patient (Figure 5 g) and h)).

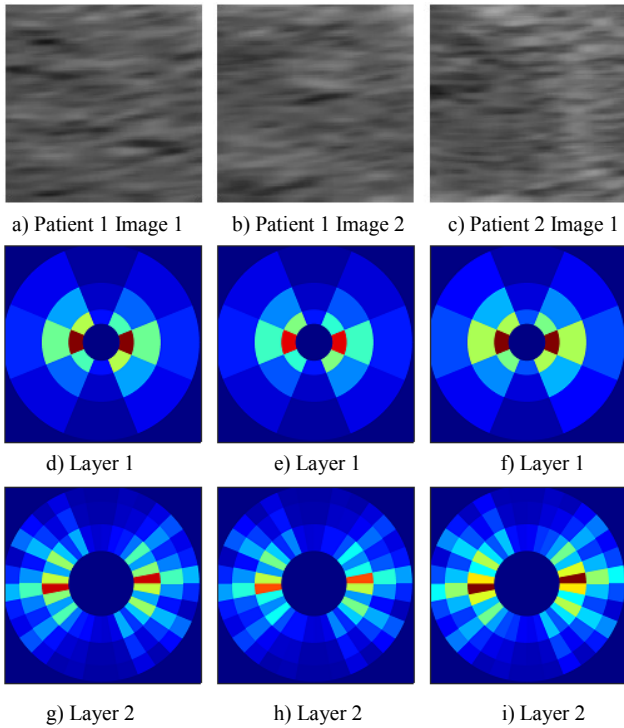


Figure 5. Three ultrasound images of a clot taken from two different patients; the first and the second layers of the scattering coefficients are represented in the frequency plane.

During this simulation, the wavelets used in the scattering operator are coming from the Morlet wavelet family. These wavelets are composed of a complex exponential multiplied by a Gaussian window. The results are not very satisfactory, so we try another wavelet family named Shannon or sinc wavelet. However, the scattering coefficients of images from two different patients still seem to be difficult to distinguish.

CONCLUSION

Multiple factors can cause a deep venous thrombosis: stasis, endothelial injury or/and hypercoagulability. This disease is especially dangerous because it may be asymptotic and create a pulmonary embolism. In our project, we aim at characterizing the blood clot structure in order to date it,

explain its formation and estimate the risk of PE. In this paper, we create a database of ultrasound clot images and apply the scattering operator. This algorithm, based on wavelet transforms, has promising results on image classification. Nevertheless our simulation shows that the scattering operator is suffering from many drawbacks and it is not well adapted for clot characterization purposes. In future work and after having tried out other scattering parameters and wavelet families, the possibilities of statistical methods applied on our ultrasound images will be explored. We will also create groups of patients with the same pathophysiology and compare their ultrasound images using the scattering operator and statistical methods.

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