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*Research Article*

# Study On The Biomechanical Properties Of Rabbit Venous Arterialization.

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## Abstract

**Objective:** To investigate the mechanisms underlying restenosis following coronary artery bypass grafting using bridging veins.
**Design:** We established an external jugular vein bypass grafting model using rabbit carotid arteries and conducted vascular biomechanical experiments (pressurisation and stretching).
**Place & duration of study:** Our research was conducted at the Medical Animal Experiment Center of Hebei University (From 1/6/2018 to 31/12/2023).
**Methodology:** We established a rabbit model of venous arterialisation, by transplanting veins into the arterial system as bridging vessels and investigated vessel tensile mechanical and histomorphological properties.
**Results:** Control vein elasticity (k = 16.20) was less than that of the control artery (k = 58.04; P < 0.05), and vein walls were thinner. Following venous arterialisation, proliferating cell nuclear antigen and alpha-actin were upregulated and vein walls thickened (P < 0.05), with elasticity after venous arterialisation (k = 86.26) significantly higher than that of control veins (P < 0.05).
**Conclusion:** This indicates that venous intima is damaged by high pressure following arterialisation, resulting in gradual restenosis, with thickening of the venous intima and an increase in vessel elasticity. Clinically, there is potential to repeat these experiments to determine the elastic extremum of the great saphenous vein and control the pressure in the lumen of this vessel, to ensure minimal damage to the intima before anastomosis, thereby facilitating improvement of long-term patency rates following vein bridge surgery. Whether the increase in venous bridge elasticity after venous arterialisation can be controlled, with the aim of preventing early-stage restenosis, warrants investigation.

**Keywords:** Coronary artery bypass grafting; Venous arterialisation; Restenosis; Elasticity; Biomechanics.

## INTRODUCTION

Coronary atherosclerotic heart disease (also known as coronary heart disease, CHD) is one of the most common diseases of the cardiovascular system and a severe threat to human health and wellbeing. For patients with left main coronary artery disease and coronary disease affecting three vessels, coronary artery bypass grafting (CABG) is widely recognised as a surgical treatment for CHD with clear clinical efficacy [1].

In CABG, the internal thoracic, radial, and gastroepiploic arteries are all potential conduits that allow for a high blood flow patency rate in the medium-to-long term after surgery [2–4]; however, their limitations, in terms of number, length and accessibility, have largely hindered the wider application of artery conduits, while the great saphenous vein is both of sufficient length and easy to harvest, making it an indispensable option for grafts in CABG. Nevertheless, when great saphenous vein grafts are used, post-operative restenosis is much more likely to occur than when artery grafts are applied [5], significantly impacting CABG outcomes. After a vein conduit has been grafted into the arterial system, its vascular histological features and physiological functions adjust in response to hemodynamic changes, affecting the long-term patency rate of the blood conduit [6].

Based on the basic surgical methods of CABG,we generated

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an animal model of cervical vein-arterial graft (venous arterialisation) in rabbits. After modelling, arterial blood flows through the venous vascular bridge, so that the venous bridge is within the arterial blood environment. Using this model, we conducted in vitro biomechanical experiments. Although there are some differences between rabbits and humans in anatomical morphology, structure, and haemodynamics, the basic principle of using a vein as a bridge vessel to assist the transport of arterial blood under high pressure has consistent characteristics in both species, providing a scientific rationale for establishment of the model.

Currently, the majority of research into the patency of vascular bridges after CABG has a primarily clinical focus, while there are relatively few interdisciplinary research studies. Here, we explored the differences in the biomechanical parameters between veins and arteries before and after vein grafts and investigated the relationships among biomechanics, vein arterialisation, and restenosis of vein grafts. Our data provide an experimental basis for further investigation of the relationship between the biomechanical properties of autograft vessels in patients with CHD and the patency of vascular bridges after CABG.

## 2. MATHODOLOGY

2.1 We established an external jugular vein bypass grafting model using rabbit carotid arteries and conducted vascular biomechanical experiments (pressurisation and stretching). The establishment of rabbit animal model was carried out in the standardized animal medicine laboratory, and the animal experiment center of Hebei university Medical college was in charge of the rabbit feeding after the modelling. All experiments were carried out under general anesthesia in rabbits. Finally, the rabbits were killed by injecting air into the ear margin under general anesthesia condition. (the principle of euthanasia). Our animal experiment scheme used in the research was reviewed and approved by the Animal Welfare and Ethical Committee of Hebei University(AWEC), and conformed to the principles of animal protection, animal welfare and ethics, as well as the relevant national regulations for experimental animal welfare ethics. Animal use permit: SYXK-2017-002. Approval Number: 2017011.

2.2 Male rabbits (n = 24; 3.0 ± 0.5 kg) were obtained from the Laboratory Animal Center of the Medical School, Hebei University and randomly divided into two groups: experimental and control (n = 12 per group). Rabbits had free access to water. Food was prohibited for 12 h before surgery. A myograph system was provided by Professor Huo Yunlong's team, Beijing University College of Engineering. Other equipment included: a vertical oxygen cylinder (Huandong), rotameter (Zhenxing), thermostatic water bath (Taisite), small peristaltic pump (Shenchen), mercury sphygmomanometer (Yuyue), a computer connected to a stereomicroscope (Nikon) and camera (Canon). In addition chemicals for preparation of HEPES-PSS solution were from Damao Chemical Reagent Factory, α-actin monoclonal antibody was from US Dako, StreptAvidin-Biotin Complex (SABC) Staining Kits from Boster, experimental surgical instruments for small animals from Function Laboratory of the Medical School, Hebei University, and 5-0 prolene sutures and a medical micro needle holder were from Suzhou Xiehe Medical Equipment Factory.

2.3 Rabbits in the experimental group received general anaesthesia and heparinisation (ethyl carbamate 4 ml/kg, 1% heparin 2 ml/kg) injected into a marginal ear vein, and were then fixed on a sterile operating table for small animals with the part of the neck requiring surgery fully exposed. Following skin preparation and standard disinfection, a 7–8 cm incision was made at the midpoint of the anterior region of the neck, and the subcutaneous tissue, fat layer, and muscle layer blunt dissected to expose 5–6 cm of the right carotid artery and the right external jugular vein. Next, lateral vascular anastomosis of the carotid artery and external jugular vein was conducted under a 2.5× portable magnifying glass, using 5-0 prolene sutures (taking 5 mm as the standard anastomotic length). The external jugular vein graft was ligated to the two stoma at both ends and the carotid artery ligated in the middle of the stoma with 6-0 sutures, which allowed the blood from the proximal artery to flow into the distal end via the vein conduit. After confirmation that blood flow was smooth and that there was no bleeding around the stoma, the surgical incision was closed, layer by layer. After regaining consciousness, rabbits were transferred to the Laboratory Animal Feeding Center of the Medical School, Hebei University. Three days after surgery, rabbits were administered penicillin injections, to prevent infection, and underwent small animal ultrasound, to monitor blood flow patency.

2.4 Four weeks after surgery, the arterialised venous conduit was excised and divided into three segments. Segment 1, a 5 mm vascular ring, was fixed in preservation fluid for haematoxylin and eosin (HE) staining(10% Formaldehyde Solution, at room temperature). Segment 2, also 5 mm, was fixed and stored(preserve liquid as before), for immunohistochemical analysis. Finally, the third segment (approximately 3 cm long) was immediately stored in perseveration solution once excised for subsequent mechanical testing(0.9% Normal Saline). In the control group, 3 cm segments of carotid arteries and external jugular veins were excised from untreated rabbits, then HE staining, immunohistochemical analysis, and mechanical tests conducted in the same way as described for experimental animals.

2.5 The specimens were embedded in paraffin and sectioned into sections with a thickness of 5μm. The specimens were routinely dewaxed to water.Diluted with a-actin .(1:200 dilution USA Dako company )Conventional SABC staining was finished for monoclonal antibody PCNA (1:100 dilution USA Dako company), and hematoxylin restaining.Four high power fields of view for each specimen were randomly chosen for observation of the number of proliferating cell nuclear antigen (PCNA)-positive cells and corresponding total cells and the cell proliferation index calculated as follows: Cell proliferation index = PCNA-positive cell number/total cell number × 100%

2.6 The myograph was configured with its two needles inserted into blood vessel fragments at each end and 4-0 sutures used for ligation. The position and distance of the needles were adjusted to maintain the vessel in the horizontal plane, with no stretching, overlapping, or twisting, and the air remaining in the system expelled using the principle of fluid dynamics. First, the active mechanical properties of the blood vessel were measured. Calcium and potassium ions can both cause vasoconstriction. Calcium ions combine with receptor proteins inside the cytosol, causing vasoconstriction and, when potassium ion concentration outside the cell increases, this may also cause vasoconstriction, due to the activity of voltage-sensitive calcium channels. Therefore, to measure their active vascular mechanical properties, vessels were bathed in HEPES-PSS solution containing a high concentration of potassium ions, while measurement of passive vascular mechanical properties was conducted using HEPES-PSS solution without calcium for stimulation, pressurisation, and mechanical stretching tests; detailed HEPES-PSS solution compositions are provided in **Table 1**.

**Table 1.** High potassium & Calcium-Free HEPES-PSS (pH 7.4) solution components per litre of distilled water.

| Chemical Component | High potassium HEPES-PSS Quantity (g) | Calcium-Free HEPES-PSS Quantity (g) |
|---|---|---|
| $CH2OH(CHOH)_4CHO$ | 0.9900 | 0.9900 |
| KCl | 10.5900 | 0.3500 |
| NaCl | 0.2750 | 8.2990 |
| HEPES $C_8H_{17}N_2NaO_4S$ | 0.7000 | 0.7000 |
| HEPES $C_8H_{18}N_2O_4S$ | 0.7150 | 0.7150 |
| $MgSO_4$ | 0.1408 | 0.1408 |
| (anhydrous) $CaCl_2$ | 0.4107 | 0.0000 |

Each blood vessel was fully immersed in high potassium HEPES-PSS solution, the pressure adjusted to 0 kPa (20 mmHg = 2.66 kPa), and the background set using the computer attached to the stereomicroscope and Canon camera. The initial length of the blood vessel (without stretching or relaxation) was first marked and recorded. Then, blood vessels were stretched to 1.3 times their initial length by adjusting the control arms of the two myograph needles and the rotameter applied to increase the pressure within the system to 23.94 kPa, followed by a decrease back to 0 kPa, while all the other experimental parameters were maintained. The pressurisation and depressurisation procedure was repeated 10 times, to minimise internal stress. Next, mechanical tests were initiated.

The internal pressure of the system was adjusted to 2.66 kPa using the oxygen cylinder, tube, and rotameter. Blood vessel diameters were measured 5 min after stabilisation (monitored by computer). After an initial data recording, the pressure was further raised to 5.32 kPa and the vessel diameter change noted. Then, pressure was raised stepwise by 2.55 kPa each time, and changes in the vessel recorded following each increase in pressure, to a maximum of 26.60 kPa, when a depressurisation procedure was initiated. Starting at 26.60 kPa, pressure was decreased stepwise by 2.66 kPa per step, with vessel diameter recorded after each step, until the internal pressure of the system returned to 0 kPa.

On completion of the 1.3-stretch-ratio pressurisation and stretch test, the internal system pressure reduced to 0 kPa and the blood vessel was allowed to rest for 15 min to fully release the remaining internal stress. Next, the same method was applied to stretch the blood vessel to 1.4 times its original length. Again, 10 rounds of the pressurisation and depressurisation procedure were conducted and data recorded, as described above. When measurements were complete, the chamber containing high-potassium HEPES-PSS was thoroughly cleaned before replacing the solution with calcium-free HEPES-PSS.

Blood vessels were completely rested in calcium-free HEPES-PSS solution and, when they were fully diastolic, the same method that was applied to measure active mechanical properties (described above) was used to evaluate passive mechanical properties. After all procedures were completed, a 3 mm long vascular ring was cut from the remaining blood vessel and incubated without manipulation in calcium-free HEPES-PSS solution for 30 min to eliminate internal stress. Subsequently, the thickness, size, and stretch angle after full extension of the vessel were determined.

HE staining outcomes and the immunohistochemical results from arterial and venous vessels. Outer diameter changes in

rabbit blood vessels in response to changes in pressure in the experimental and control groups.

2.7 All data were statistically analysed using SPSS 25.0 statistical software. Measurement data are presented as mean ± standard deviation. The significance of differences between groups were evaluated using the t-test, with a significance level of 0.05.

## 3.RESULT

### 3.1 Vessel morphology

All 24 rabbits survived the experiment and, in the experimental group, all vein conduits provided smooth blood flow. Venous conduits were removed 4 weeks after surgery, HE stained, and analysed immunohistochemically alongside normal carotid arteries and external jugular veins, as controls. We found that: 1) Arteries had thicker walls than veins; however, the tunica media and intima of the vein grafts appeared to thicken after grafting (Figure 1, 2), with their inner diameters decreasing after grafting. 2) Immunohistochemical analysis revealed increased staining for α-actin in the tunica media and neointima of veins from rabbits in the experimental group compared with control veins, with deeper coloured staining and high levels of PCNA expression. These data confirm that smooth muscle proliferates following venous arterialisation, while the venous tunica media-intima thickens.

**Figure 1.** Optical micrograph of a normal rabbit arterial vessel cross-section stained with haematoxylin-eosin. Magnification, 40×

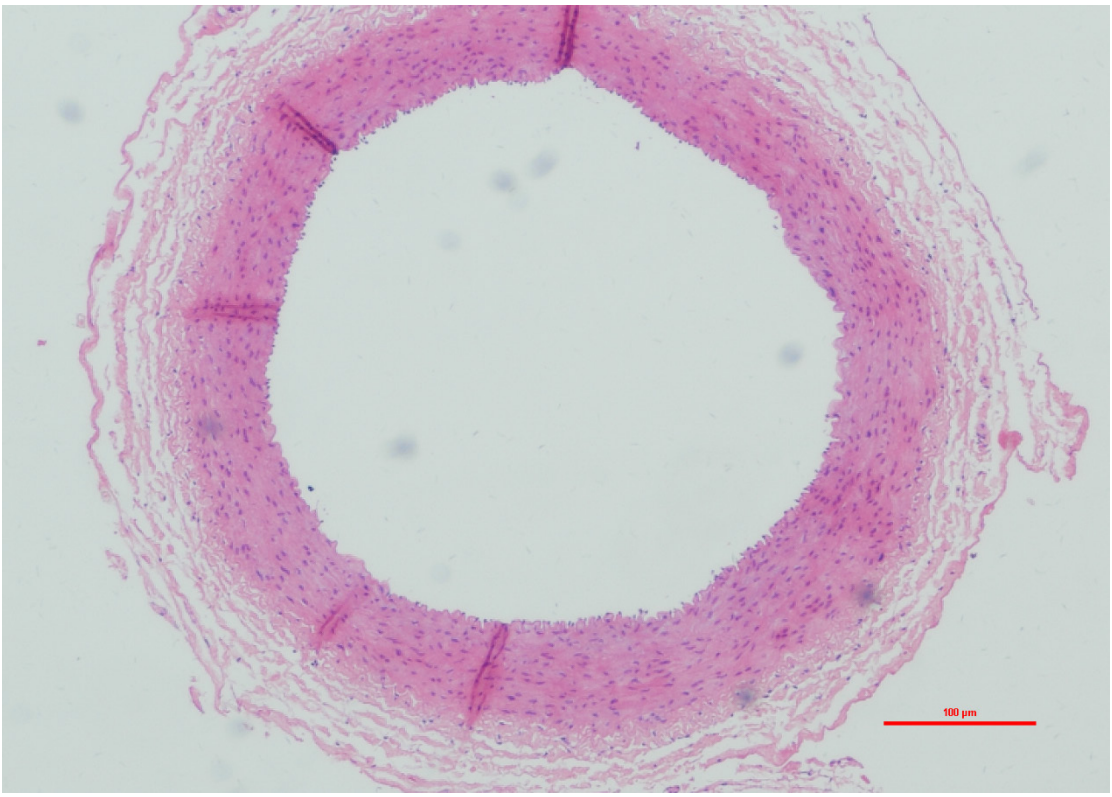
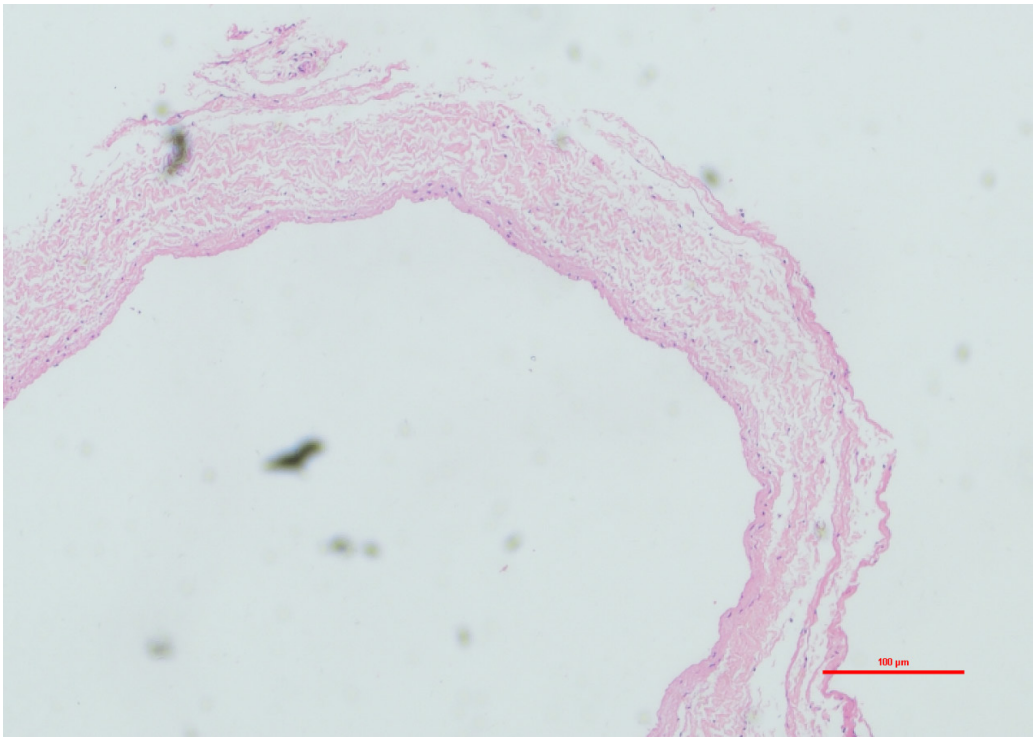


**Figure 2.** Optical micrograph of a normal rabbit venous vessel cross-section stained with haematoxylin-eosin. Magnification, 40×.




### 3.2 Pressurisation and mechanical stretch analysis

#### *3.2.1 Effect of pressure on blood vessel diameter and wall thickness*

As pressure in the vascular lumen gradually increased from 0.00 to 26.60 kPa, the outer diameters of blood vessels in all the three groups, determined by both visual observation and computer analysis, increased to a certain value, then gradually reduced, and finally stabilised around a specific fixed value, depending on the experimental group: venous experimental group range, 5085.64 to 10338.66μm(Mean values of 4 groups of data) (**Figure 3**); venous control group range, 4796.14 to 10606.89 μm (Mean values of 4 groups of data) (**Figure 4**); arterial control group range, from 4012.19 to 5658.90 μm(Mean values of 4 groups of data) (**Figure 5**). Smooth muscle cells and elastic fibres in blood vessel walls both protect the vessels and control their elastic deformation within a reasonable range; that is, these features restrict the excessive expansion of blood vessels. In addition, as the outer diameter increased, the thickness of the vessel walls gradually decreased, and finally stabilised at a specific value, which reflected the inherent thickness of the material constituting the blood vessel wall (i.e., smooth muscle cells and elastic fibres) (**Fig. 3**).

**Figure 3.** Changes in the experimental group vessel outer diameter according to pressure variation under different stretch ratios and active and passive states (n = 10; measurements in μm)

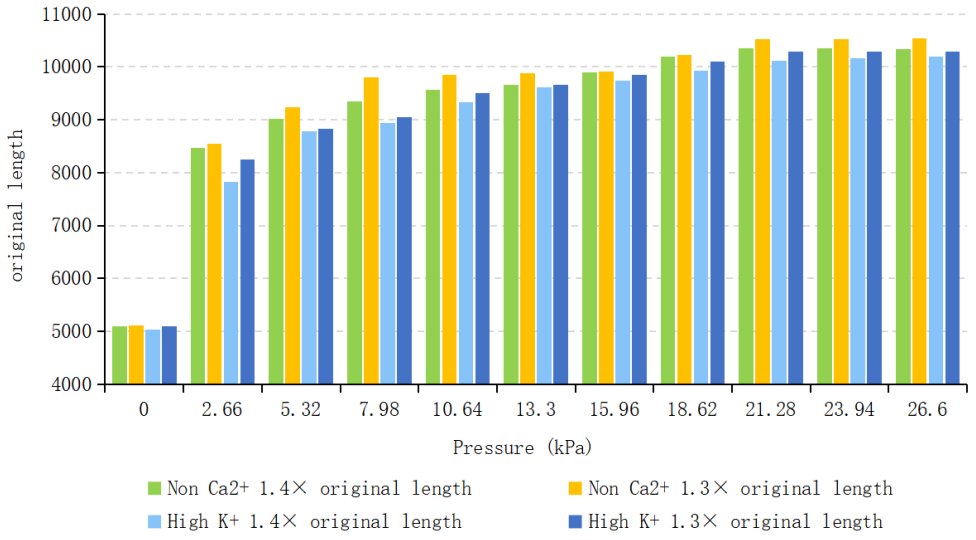


**Figure 4.** Changes in control group venous outer diameter according to pressure variation under different stretch ratios, active and passive states (n = 10, measurements in μm)

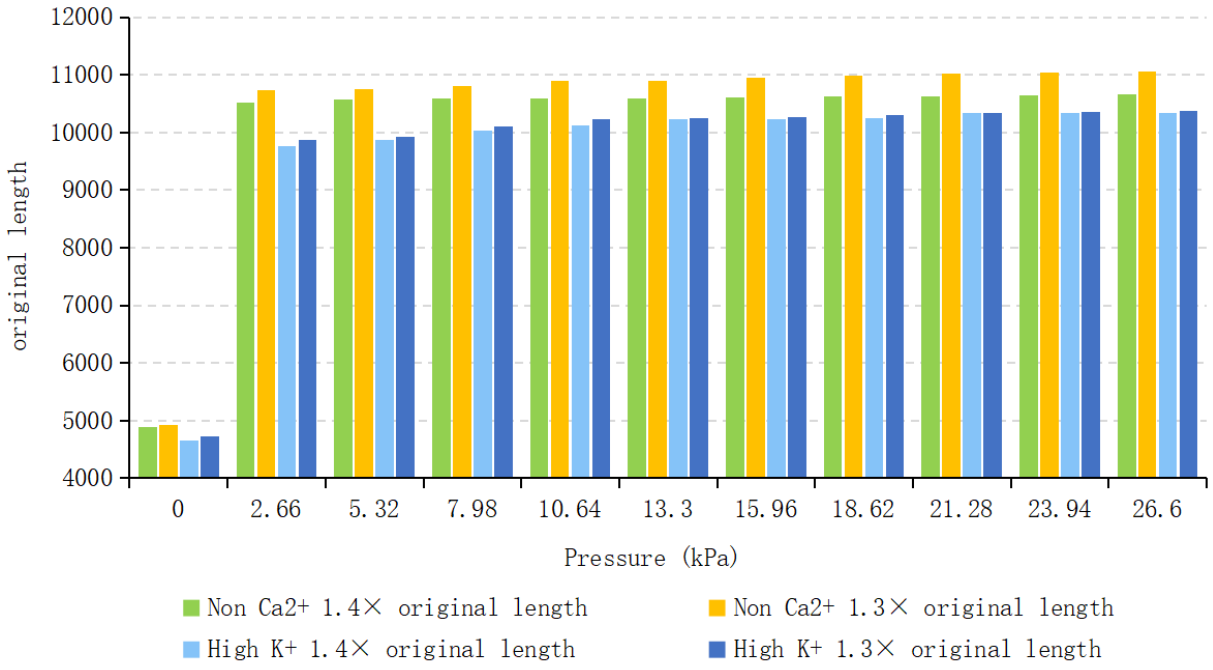



**Figure 5.** Changes in the control group arterial outer diameter according to pressure variation under different stretch ratios, in active and passive states (n = 10; measurements in μm).

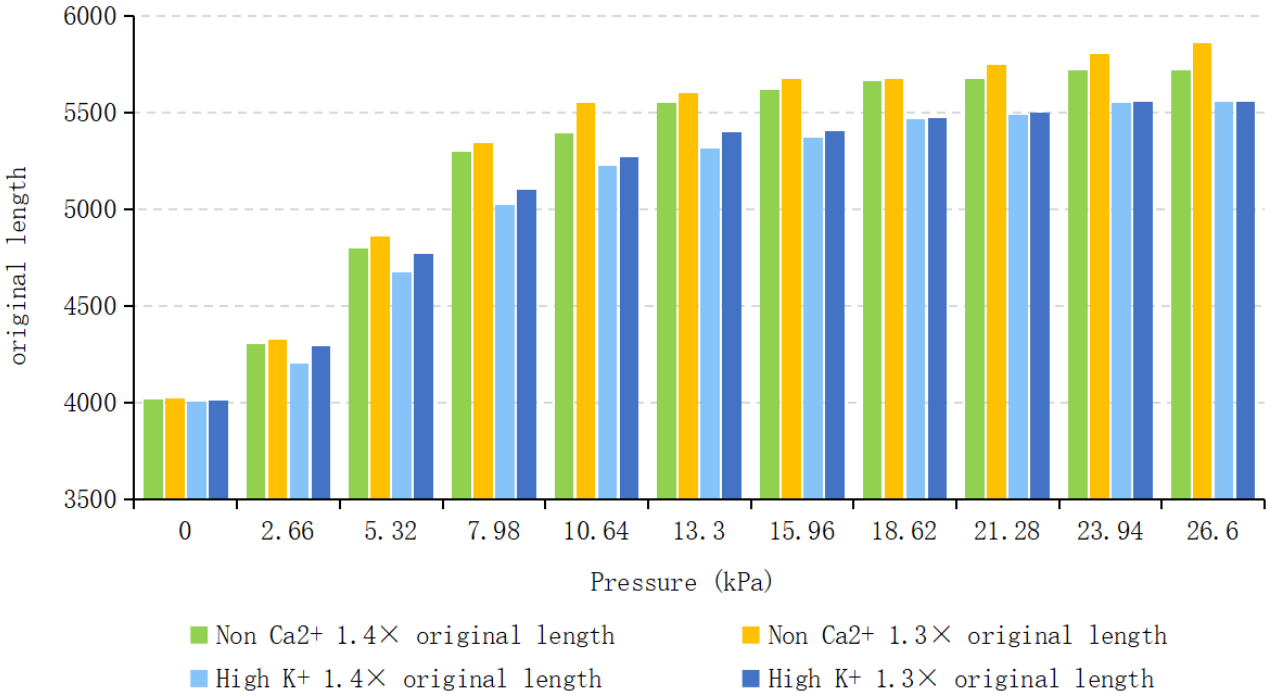
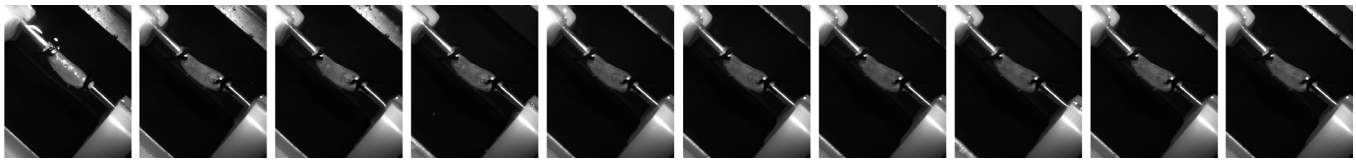


**Figure 6.** Real-time photographs of venous bridge vessels from the experimental group as intravascular pressure increased (pressure range from left to right: 2.66–26.60 kPa)



***3.2.2 Variation in control vein diameter under different tensile ratios and in response to high potassium and low calcium***

For each type of blood vessel (control veins, control arteries, and arterialised veins), as intravascular pressure increased, the outer diameter first increased to the vicinity of the elastic extremum, and then fluctuated around a defined value; however, there were subtle differences in the characteristics of these changes in response to alteration of the tensile ratio applied to the blood vessels and the chemical environment in which they were placed. **Figure 7** shows the effect of different tensile ratios on control vein outer diameters in a passive environment (perfused with HEPES-PSS lacking $Ca^{2+}$). As the pressure rose above 5.32 kPa, the outer diameters of the two groups began to differ, with the venous outer diameter at a tensile ratio of 1.4 smaller than that at a ratio of 1.3. Further, as pressure increased, the difference in the outer diameter also gradually increased ($P < 0.05$). As shown in **Figure 8,** under the same tensile ratio (1.4) control veins suffused with different solutions (high K+ or low $Ca^{2+}$) also exhibited differences in diameter. Again, the difference was apparent at pressures above 5.32 kPa, with the external diameter of the vein in the active state (high $K^+$ solution) smaller than that in the passive state (low $Ca^{2+}$ solution); however, in this comparison, the difference between the external diameter in the two gradually decreased with increasing pressure ($P < 0.05$).

**Figure 7.** Curve showing changes in the mean outer diameters of rabbit veins from the control group under different tensile ratios as intravascular pressure increased.

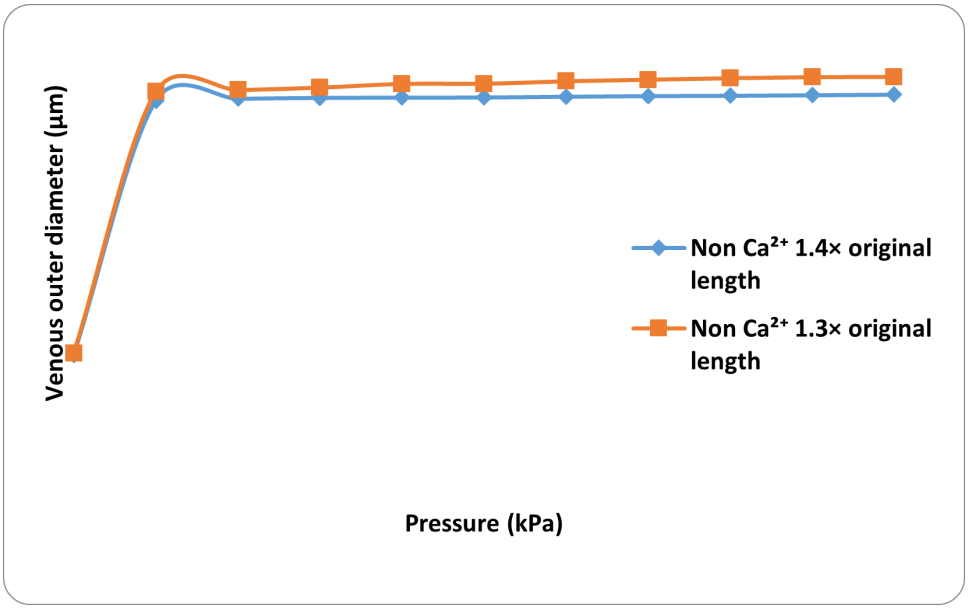
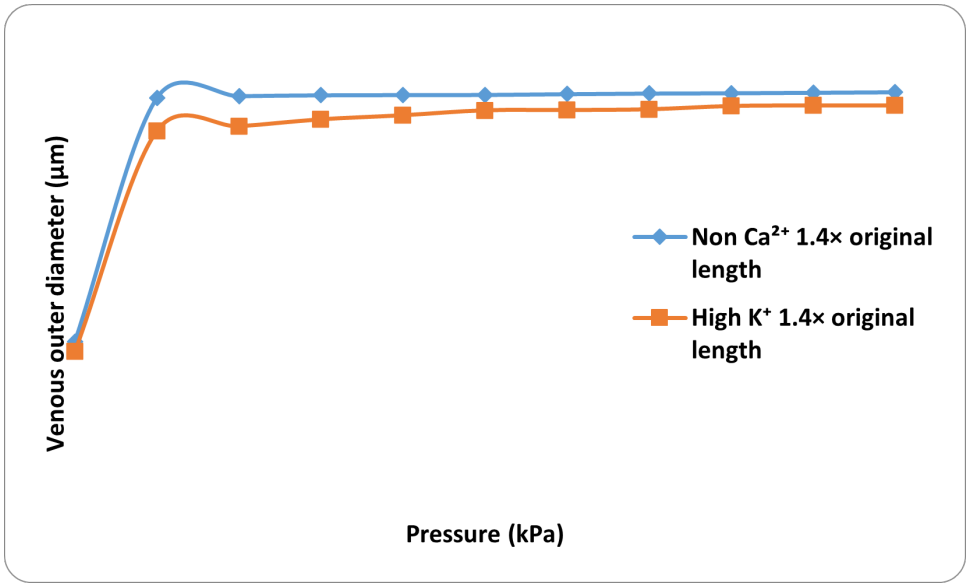


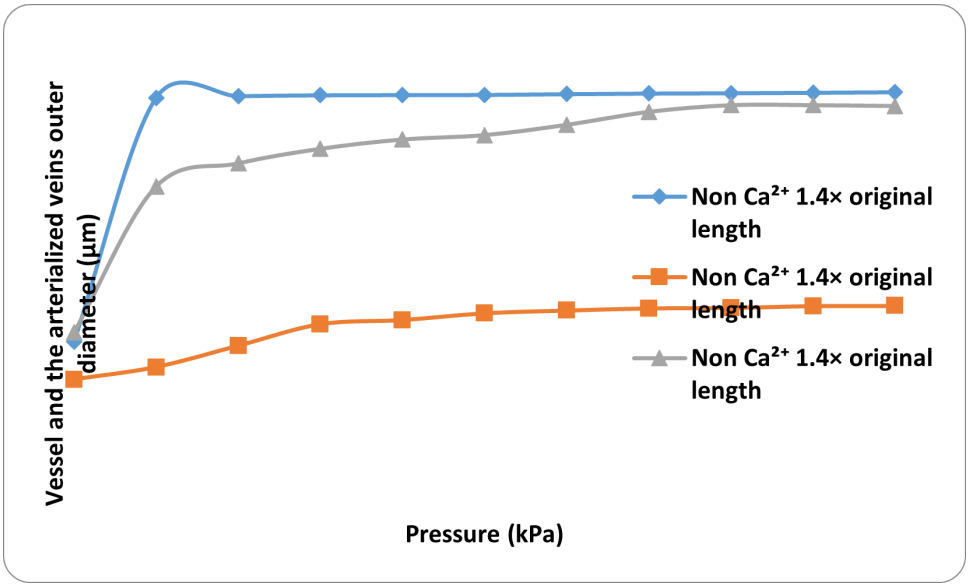
**Figure 8.** Curve showing changes in the mean outer diameter of control group rabbit veins in the active (high potassium) and passive (no calcium) states as intravascular pressure increased.




### *3.2.3 Comparison of the effects of intravascular pressure on veins and arteries*

Evaluation of the differences in the mechanical parameters of arteries and veins in the control group demonstrated that the outer diameter of both types of vessel was similar before initiation of the experiment. On commencement of compression and stretching, the outer diameter of the vein initially increased rapidly, and stabilised when the intravascular pressure rose to 5.32 kPa. For arteries, the outer diameter stabilised when the intravascular pressure rose to 7.78 kPa. Once stabilised, the external diameter of the vein was almost twice that of the artery under the same conditions ($P < 0.05$). In addition, although the changes in both types of vessel were consistent with the overall alterations in pressure, as intravascular pressure increased, veins reached their elastic limit more quickly, and their outer diameter increased significantly less than that of arteries. The slope (k) was calculated as the increase in the outer diameter (ΔR)/pressure change (ΔP), with values for vein and artery of k = 16.20 and 58.04, respectively, indicating that the elasticity and compliance of the vein after contrast were smaller than those of arteries ($P < 0.05$). These data indicate that long-term stretching may impair the structure and function of the venous intima (Fig. 6).

**Figure 9.** Curves showing changes in the mean outer diameters of rabbit veins and arteries in the control group and veins in the experimental group in the passive state (no calcium) and under a tensile ratio of 1.4, as intravascular pressure increased. (The red represents the change trend of arteries in the control group. Blue represents the change trend of veins in the control group.Green represents the change trend of veins in the experimental group.)



### *3.2.4 Comparison of the effects of intravascular pressure on arterialised and control veins*

Comparison of the mechanical parameters of veins in the experimental group (after arterialisation) with those in the control group demonstrated that, as the intravascular pressure increased, the diameter of the arterialised vein before the external diameter reached a constant value (ΔR) was significantly greater. In the control group, at intravenous pressures > 5.32 kPa, the outer diameter of the vein increased within a small range; however, in vessels after arterialisation, it increased steadily, and did not begin to stabilise until 15.96 kPa. Further, following venous arterialisation veins had significantly greater elasticity (k = 16.20 vs. 86.26), and their walls became thicker ($P < 0.05$). The mechanisms underlying these changes may involve smooth muscle proliferation and "hypertension adaptation". [7]Changes in outer diameter in response to increasing pressure under different tensile ratios and in the active or passive states for control veins, control arteries, and arterialised veins are presented in Figures 10, 11, and 12, respectively.

**Figure 10.** Curve showing changes in the mean outer diameter of rabbit veins from the control group in the active (high potassium) and passive (no calcium) states under different tensile ratios, as intravascular pressure increased.

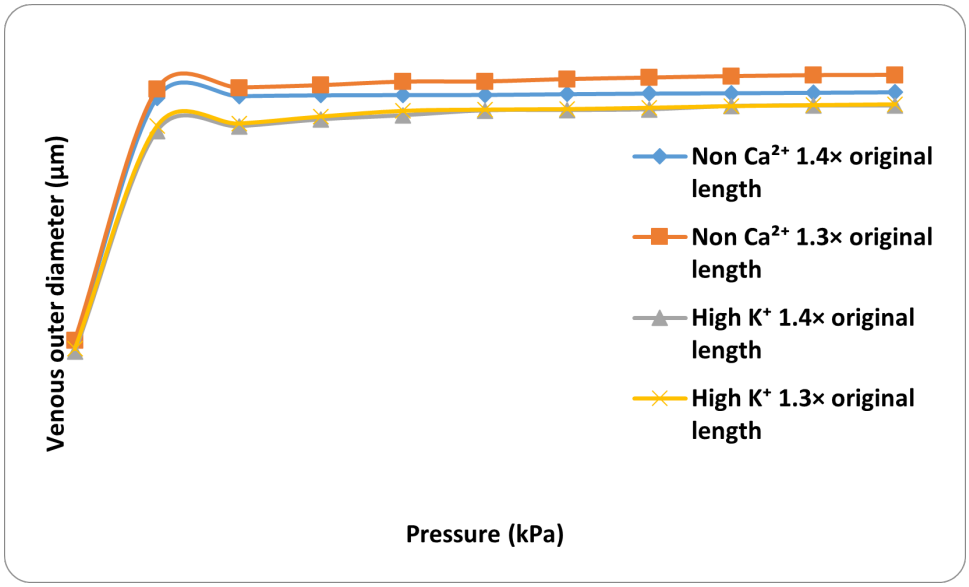



**Figure 11.** Curve showing changes in the mean outer diameter of control group rabbit arteries in the active (high potassium) and passive (no calcium) states under different tensile ratios, the as intravascular pressure increased.

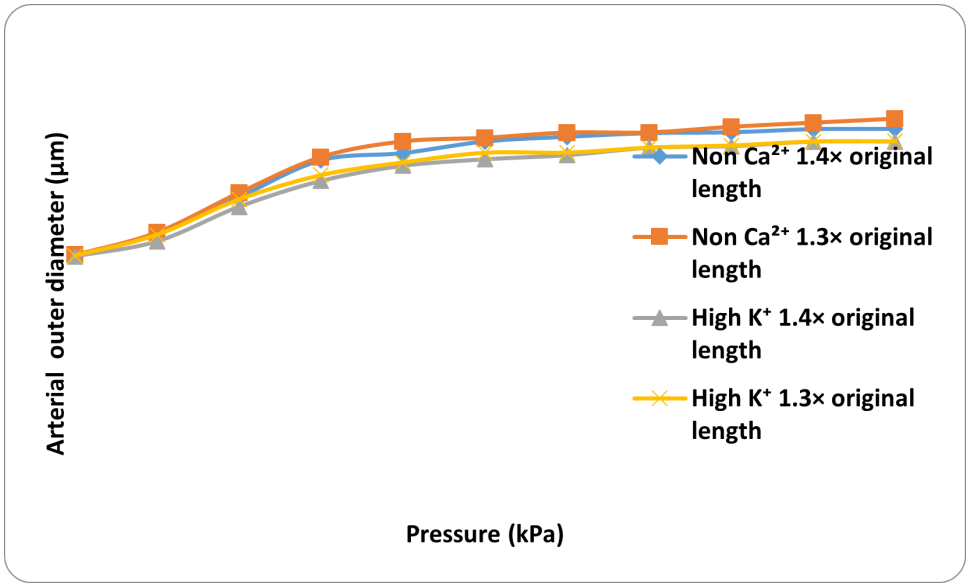
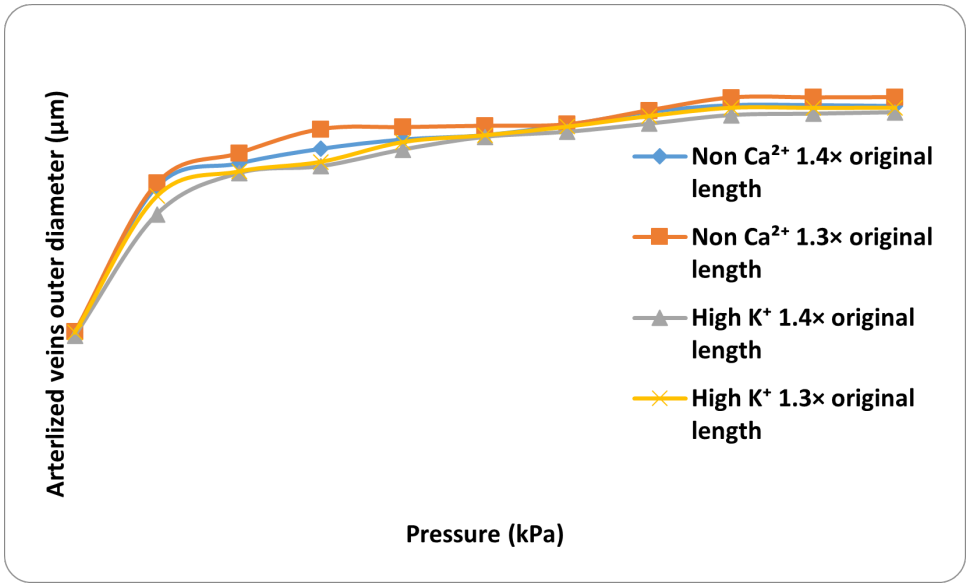


**Figure 12.** Curve showing changes in the mean outer diameter of experimental group rabbit veins in the active (high potassium) and passive (no calcium) states under different tensile ratios, as the as intravascular pressure increased.




## 4.DISCUSSION

In terms of the anatomical and physiological basis of blood vessels, the thickness of the venous wall is significantly smaller than that of the arterial wall, and the arterial wall is rich in collagen, elastic fibers, and smooth muscle cells, which provides histological and physiological basis for maintaining arterial elasticity and the ability of withstanding higher blood pressure. The HE staining of the cross-section of our rabbit blood vessels further confirmed these (Figure 1, 2).

The data presented in Figure 6 demonstrate that, as the intravascular pressure rises, the outer diameter of the blood vessel gradually increases and eventually oscillates around a certain constant value. Although the change in the outer diameter is difficult to observe using the naked eye, it is easy to detect and quantify subtle these variations using a stereo microscope connected to a digital camera and a computer. We found that the outer diameter of blood vessels first increased as intravascular pressure rose, subsequently stabilising at a specific constant value. Further, our data demonstrate that diameters varied according to tensile ratio under the same pressure, with smaller diameters at the higher tensile ratio. Mechanically, this phenomenon can be explained by the fact that, as circumferential forces increase, due to stretching, they result in a smaller change in the outer diameter of the blood vessel. Moreover, under the same pressure and tensile ratio, the outer diameter of blood vessels in the active state were smaller than those in the passive state, which is attributable to the active contraction of smooth muscle in the blood vessel wall in response to high potassium stimulation. In addition, as the pressure in the lumen increases, it can be inferred from our data that the state of the vasculature (including but not limited to stretching ratios) has more influence on morphological changes in blood vessels (including but not limited to the external diameter) than the external environment, according to the consequences of different tensile ratios on variation in blood vessel outer diameter in different mechanical states (active and passive); however, further discussion is needed, as shown in Figure 7 and 8.

Changes in the outer diameter of vessels in response to increasing pressure in the experimental and control groups under the same conditions are plotted in Figure 9. Before initiation of vessel compression and stretching, the outer diameters of these groups of vessels were all similar. Once intravascular compression commenced, the outer diameter of veins initially increased rapidly, then stabilised at almost twice that of arteries under the same conditions. In addition, although the overall trend in arteriovenous changes with pressure was the same in arteries and veins, as intravascular pressure increased, veins reached their elastic extremum more rapidly, with the increase in their outer diameter significantly less pronounced than that of arteries, indicating that the elasticity and compliance of veins are lower than those of arteries. This is a consequence of the thinner venous wall, and its lower content of smooth muscle cells, and collagen and elastic fibres, as well as the physiological “rigidity” of these blood vessels. Comparison of differences in the mechanical properties of control veins and veins collected after venous arterialisation in rabbits indicated that, after transplantation into the arterial system, venous elasticity increased compared with that of control veins. These findings were supported by the evaluated mechanical parameters and could also be detected by examination of histological features.

After venous arterialisation, the long-term hypertensive

environment leads to early damage of the vein intima; hence, smooth muscle cells migrate to the intima, causing it to thicken, and promote lumen restenosis. Further, the increase in the number of smooth muscle cells in the intima may be the physiological mechanism underlying the increase of vascular elasticity in vein bridges[8]. But few studies have focused on treatments targeting smooth muscle cell proliferation and intima thickening in the middle stage of restenosis after surgery. Especially by combining molecular biology, hemodynamics, pathophysiology, and mechanics research, exploring the mechanical mechanisms of vascular histological changes, and use mechanics theory to understand or intervene in vascular endothelial injury and smooth muscle proliferation, and explore possible clinical solutions, will positively influence efforts to prevent restenosis of conduits (especially venous conduits) in the middle-late stage of restenosis after CABG [9–10]. This is also one of the important innovations of our study.

The tunica adventitia of vessels comprises relatively loose connective tissues [11–13] and has a decisive role in vascular biomechanical characteristics [14-15]. The basic attributes, including volume, proportion, structure, and shape of the collagen and elastic fibres and smooth muscle cells, further influence the biological characteristics of the tunica media. Collagen fibres of venous and arterial vessel walls share the same basic components, but are differentiated by their volume, with arterial walls thicker, as they contain more elastic and collagen fibres than venous walls. After grafting into the arterial system, veins are subjected to the mechanical effects of relatively high blood pressure and fast blood flow, which harm the venous tunica intima and force smooth muscle cells from the tunica media layer to move into the intima layer. Under these circumstances, smooth muscle cells proliferate and thicken the intima layer, leading to blood vessel restenosis [16–18].

Our mechanical experiments revealed that, among control vessels, veins presented with lower elasticity and compliance than arteries, indicated by the fact that, with increasing pressure, venous outer diameters exhibited smaller increases before rapidly reaching a constant value. When veins are grafted into the arterial system, they bear constantly high blood pressure exerted by the blood flow in the arterial system. Previous mechanical studies have suggested that, under these circumstances, mechanical damage from different directions (e.g., normal stress and shear stress) occurs [19-20]. The possible relationship between such damage and intima thickening is an avenue for further exploration. Furthermore, when veins were arterialised, their vascular outer diameters increased more significantly in response to rising pressure than observed in control veins, likely because the smooth muscles were proliferating and the intima thickening. This indicates that the elasticity of the blood vessels improves after arterialisation, differing from the vessel stenosis led by atherosclerosis (lipid deposition, calcification, etc.) [21] Therefore, when restenosis occurs following venous grafting, the mechanical properties (wall thickness, elasticity coefficient, compliance, etc.) are enhanced relative to those before the graft. that stepwise transition from venous-to-arterial conditions results in a partial restoration of circumferential stretch and circumferential, but not axial, stress through vessel dilation and wall thickening in a primarily outward remodeling process. These remodeled tissues also exhibited decreased mechanical isotropy and circumferential, but not axial, stiffening[22].

## 5.CONCLUSION

We draw the following conclusions. 1. With increased pressure inside blood vessels, veins reach maximum elasticity more rapidly than arteries. The venous intima is particularly vulnerable to the relatively high pressure experienced in the arterial system, likely leading to damage to the intima and its function [23]. In the future, we may repeat these mechanical experiments to identify the maximum elasticity value of the great saphenous vein. It could be anticipated that, when the great saphenous vein is extracted, a pressure sensor could be used during CABG to ensure that its internal pressure remains below a maximum point, to minimise damage to the intima before vascular anastomosis, which has potential to significantly improve the patency rates in the middle-to-long term after venous grafting [24-25]. 2. After venous arterialisation, elasticity and compliance of the grafted vein rise; however, vascular stenosis caused by the intima thickening that gradually occurs due to smooth muscle proliferation, which also contributes to the pathophysiological basis of vascular restenosis. Thus, determining the relationship between vascular mechanical properties and vascular graft stenosis could facilitate control of intima thickening and the rise in elasticity parameters following venous arterialisation, positively influencing the patency rates of venous conduits after transplant into the arterial system. Our data will promote discussion of the relationship between the biomechanical properties of autologous vascular grafts in patients with CHD and vascular patency after CABG from a novel perspective[26].

## Declarations

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### Ethical Statement

The animal experiment scheme used in our research was

reviewed and approved by the Animal Welfare and Ethical Committee of Hebei University (AWEC), and conformed to the principles of animal protection, animal welfare and ethics, as well as the relevant national regulations for experimental animal welfare ethics. Animal use permit: SYXK-2017-002. Approval Number: 2017011.

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**Competing Interests**

We have no competing interests.

**Authors' Contributions**

L.T.T., Z.Y.H., N.P., Z.Z.M., N.X.L., S.W.Z and H.L.C. were responsible for the preliminary preparation of experiments and their implementation. L.T.T., Z.Y.H. and N.P. were responsible for the processing of experimental data and writing the paper. N.P. was in charge of the thesis audit. S.W.Z. was responsible for providing laboratory and experimental materials. L.F.L., Z.Y.H designed the project and the applicant for the project. Z.Y.J. was in charge of the overall evaluation. All authors gave final approval for publication.

**Availability of data and material**

The datasets related to the current study are available from the corresponding author on reasonable request.

**Preprint**

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