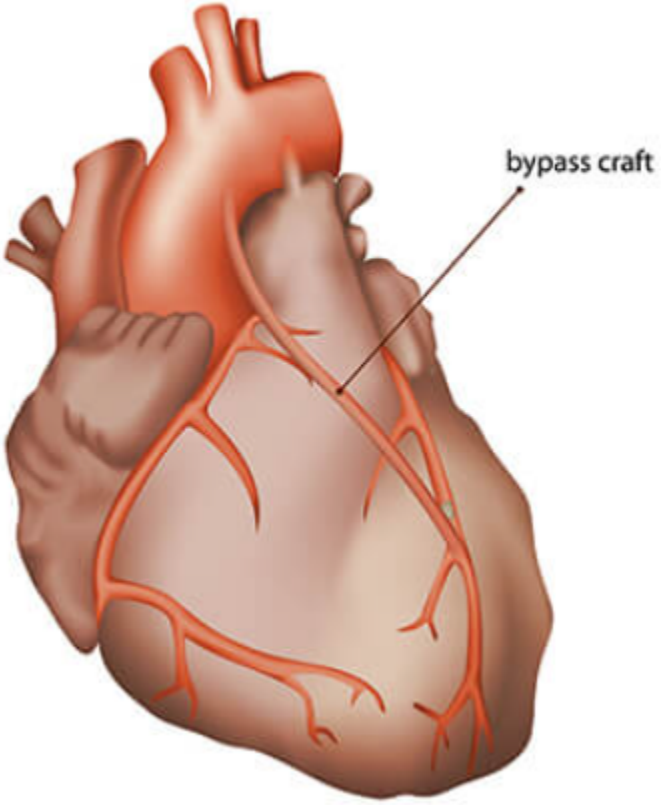
Disease state analysis

Need Statement: A way to reduce the chances of leakages due to graft failure after Coronary artery bypass graft surgery.

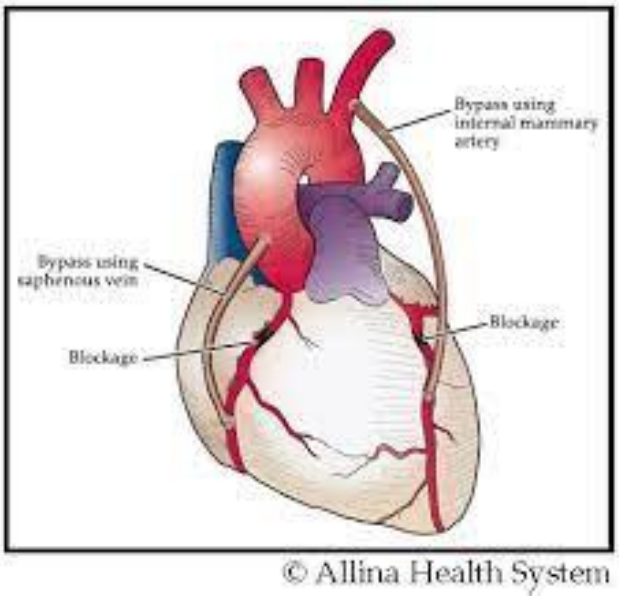
1.1

Anatomy and physiology :

The following image depicts the state of heart after bypass and before failure.



The prevalent state disease caused by CABG graft failure is the condition after bypass operation and the patient is discharged and now after few years of discharge . Patient is fully functional and can perform day to day practice.



2.1

There are no or minimal obstructions in veins as bypass has cured it.

The stent make sures that there is enough blood flow and also make sures that the cross section of the vein doesnt decreases due to swelling or fat depositon.

Pathophysiology :

How does disease affect the normal functions

Three pathophysiologic processes lead to SVG failure: thrombosis and technical failure is the predominant mechanism within the first week and during the first month after CABG, followed by intimal hyperplasia from 1 month to 1 year, and atherosclerosis beyond 1 year.

Early failure is attributed to technical (ie, graft trauma during harvesting, anastomotic deficiencies), conduit-related (ie, mismatch in conduit size or preexisting graft pathology), or extrinsic factors (ie, hypercoagulability) causing acute thrombosis. Mechanical forces and ischemia-reperfusion injury during harvesting and storage result in endothelial denudation and smooth muscle cell (SMC) damage. De-endothelialization leads to exposure of the extracellular matrix and activation of extrinsic coagulation

cascade by the tissue factor. Reduced bioavailability of prostacycline and nitric oxide (NO) lead to vasoconstriction and stasis, which further promotes fibrin accumulation, adherence of activated platelets and leukocytes on the luminal surface, and thrombus formation.

SVG intimal hyperplasia is an adaptive mechanism to high arterial pressure, a process called “arterialization,” and occurs within months after CABG. It can cause mild lumen reduction but rarely leads to significant early stenosis. Activated platelets secrete multiple cytokines (ie, interleukin-1, interleukin-6) and growth factors (ie, platelet derived growth factor, transforming growth factor beta) that promote SMC proliferation. In parallel, coagulation activation leads to thrombin formation and eventually deposition of polymerized fibrin. Thrombin stimulates SMC proliferation both directly and indirectly through PDGF secretion from platelets. Neo-endothelium begins to form from the edges of the injury zone over a layer of platelets and fibrin. Approximately 4 days after graft insertion, SMC proliferation reaches a peak, and the SMCs of the medial layer undergo phenotypic modulation from a quiescent contractile state to a synthetic stage, similar to fibroblasts, migrating to the intima. Further thickening of the intima takes place by secretion of extracellular matrix, composed of elastin, collagen, glycoproteins, and proteoglycans. High proliferative adventitial fibroblasts migrate to the intima and differentiate into myofibroblasts, contributing to intimal thickening. The aforementioned processes start initially at the anastomotic sites, expanding over time throughout the entire SVG. Innate immune system cells, such as mast cells and natural killer cells, also participate in development of intimal hyperplasia.

Clinical Presentation:

Impact , Signs and Symptoms

Ten of 24 patients (42%) with SVG failure presented with an ACS . Of the 10 patients who developed SVG occlusion, 3 met the Academic Research Consortium (ARC) .
Initial procedure was complicated by recurrent chest pain and Thrombolysis in Myocardial Infarction flow grade 2 SVG the following day requiring repeat balloon angioplasty and catheter thrombectomy (Angiojet, Possis Medical, Minneapolis, Minnesota).

We have observed restricted flow in SVG
The patients found to have an SVG occlusion presented with unstable angina (n = 1), stable angina (n = 3), or were asymptomatic (n = 3).

Table 1. Clinical Presentation of Stent Failure in the SOS Trial

Presentation	Total Patients	BMS Patients	PES Patients
ACS	10 (42%)	7 (35%)	3 (75%)
NSTEMI	7 (29%)	6 (30%)	1 (25%)
UA	3 (13%)	1 (5%)	2 (50%)
Stable angina	9 (37%)	9 (45%)	0 (0%)
Asymptomatic	5 (21%)	4 (20%)	1 (25%)

Values are n (%).

ACS = acute coronary syndrome; BMS = bare-metal stent(s); NSTEMI = non–ST-segment elevation acute myocardial infarction; PES = paclitaxel-eluting stent(s); UA = unstable angina.

One additional patient presented initially with subtotal in-stent restenosis but subsequently developed definite stent thrombosis after repeat SVG PCI. This patient presented with stable angina 12 months after randomization due to sub-total in-stent restenosis as well as a new severe stenosis more distally in an SVG supplying the right PDA. He underwent repeat PCI of both SVG lesions, and 5 days later he developed stent thrombosis.

7 patients with occluded vein grafts (without definite stent thrombosis) at follow up, 4 were still taking dual antiplatelet therapy at the time of follow-up and 3 had stopped clopidogrel before the discovery of the occluded SVG.

Clinical Outcomes

Morbidity and mortality

A limited number of studies have been published on the association of vein graft failure and clinical outcomes. A well-known study from the 1990s by Lytle et al[13] compared long-term survival in 2 cohorts: (1) 723 patients with prior CABG who had graft stenosis but did not undergo any revascularization within 12 months of follow-up and (2) 573 patients without documented graft stenosis. After a mean follow-up of 6.9 years, they found that patients with vein graft stenosis occurring within 5 years after surgery and patients with no vein graft stenosis had similar outcomes. However, patients with significant stenosis in SVGs to the left anterior descending coronary artery had higher rates of death and cardiac ischemic events. An analysis from the Duke Cardiovascular Databank reported the clinical impact of early vein graft failure in 1,243 patients who underwent angiography for clinical reasons within 18 months after CABG.[14] The investigators also found that vein graft failure was associated with death, myocardial infarction, or revascularization driven by revascularization. Recently, the results of the long-term clinical follow-up study of the PREVENT IV trial[9] were published. Saphenous vein graft failure was reported in 43% of patients (787/1,829 patients) and was subsequently associated with an increase in revascularization but not with death and/or myocardial infarction. The authors concluded that although the absolute number of failed grafts did not correlate with long-term clinical outcomes, the proportion of grafts with SVG failure did. A limitation of this analysis was that the trial was not powered for SVG failure and clinical outcome, so modest but significant correlations with outcome could have been missed. Further randomized studies are needed to further evaluate the role of SVG failure on subsequent clinical outcome.

Epidomology

Incedence and prevalence

Approximately 50% of saphenous vein grafts (SVGs) fail by 5 to 10 years post-coronary artery bypass grafting (CABG) and between 20–40% fail within the first year (1,2). While SVG failure can sometimes be silent, when symptomatic events occur, SVG percutaneous coronary intervention (PCI) is often performed.

Economic Impact

Economic related to the same

Cost of CBAG is **ranged from $44,824 to $448,038**.and around 40% of those who under go SVG will under go a failure within 10 years.Which adds up to a lot of cost.

There are more than 3.5 lakh CBAG per year alone in USA .

Resources

https://pubmed.ncbi.nlm.nih.gov/34460327/
https://jtd.amegroups.com/article/view/27659/html#:~:text=Approximately%2050%25%20of%20saphenous%20vein,(PCI)%20is%20often%20performed.
https://www.advancedcardiovascular.org/services-williamsburg/cardiac-diagnostic-treatments/coronary-artery-bypass-graft-surgery/

Index of comments

CE 208 Soil

HYDROMETER ANALYSIS & ATEMBERG LIMITS

AIM: To determine the particle size distribution characteristics of fine grained soil.

Apparatus

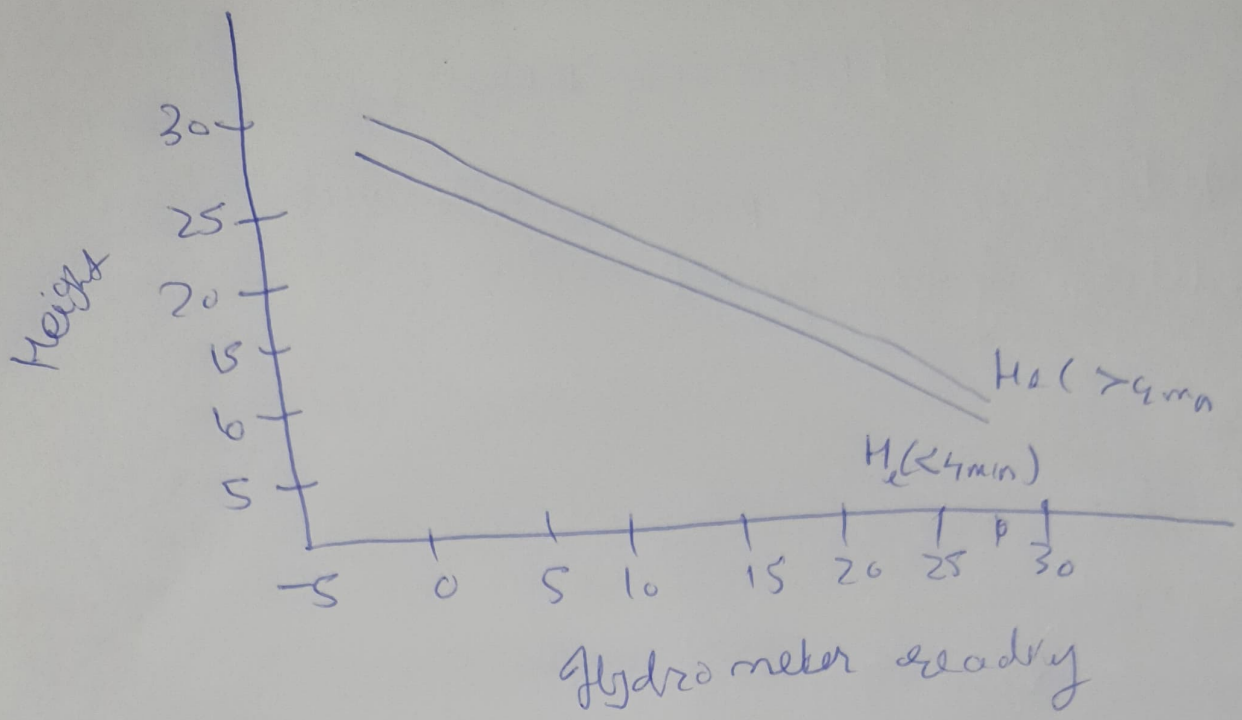
1. Hydrometer
2. Stirring Apparatus
3. Graduated Cylinder of 1000ml capacity
4. dispersing agent
5. Distilled water
6. Stop watch
7. Thermometer

Formulations

$$D = \sqrt{\frac{30\eta}{g(G_s - G_1)}} \sqrt{\frac{L_e}{t}}$$

$G_s = 2.7$ = Density of soil

$G_1 = 1$ = Density of solution



The percentage of soil remaining in suspension at level which the hydrometer is measuring the density

$$P = \frac{100000}{W} \frac{G_s}{G_s - G_1}(R_h - G_1)$$

Observation & Calculation

$W_s = 50g$

$G_s = 2.7$

$G_m = 1.05$

$\alpha = -3$

Volume of hydrometer = 80ml

Height of Jar = 3.5cm

$$A = \frac{100}{3.5} = 28.57 cm^2$$

Hydrometer reading	H	h/2	he = h + h/2	Effective head $H + \frac{1}{2}(h - $
30	0.9	8.1	9	7.6
25	2.9	8.1	11	9.6
20	4.9	8.1	13	11.6
15	6.9	8.1	15	13.6
10	8.9	8.1	17	15.6
5	10.9	8.1	19	17.6
0	12.9	8.1	21	19.6
−5	14.9	8.1	25	21.0

ATTERBERG LIMIT OF SOIL

CE208 [illegible]
BATCH I
Abhishek

Aim :- To determine liquid limit, plastic limit and shrinkage limit of fine grained soil.

Apparatus :-

1. Mechanical liquid limit device
2. Grooving tool
3. Spatula
4. Mixing dish
5. Containers for determination of moisture content
6. Balance - Sensitive to 0.01 g.
7. Drying oven
8. IS Sieve - 425 micron

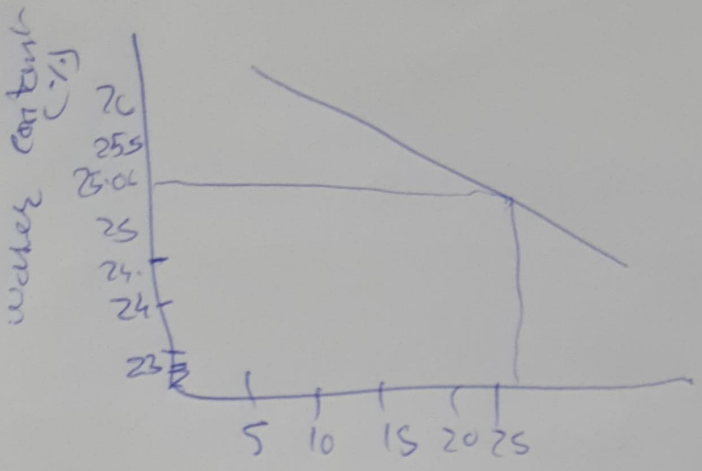
Observations & calculations

G = 2.7

Sl.No	1	2	3	4
Container No.	9	8	524	2
Number of blows	48	14	32	23
w_1 [Empty weight of container]	19.33g	20.92g	20.44	21.50
w_2 [Empty weight of container + wet soil]	24.70g	30.75g	31.50	24.52
w_3	25.85g	29.70	29.28	22.13g
% of water	23.97	25.69	25.11	24.60

$$\therefore w_c = \left(\frac{w_2 - w_3}{w_3 - w_1}\right)$$

1] $\therefore w_c = 23.77\%$



→ 25.06% is liquid limit. for 25 blows

$w_c = 25.06\%$.

Flow index

$$I_f = \frac{25.65 - 23.77}{\log_{10}\left(\frac{48}{14}\right)}$$

$$I_f = 3.59$$

Plastic limit.

Water content (%)

$$\Rightarrow \frac{28.11 - 27.62}{27.62 - 23.55} \Rightarrow 12.04\%.$$

Plastic limit

$w_p = 12.04\%$.

$I_p = 25.06 - 12.04$

$I_p = 13.02\%$.

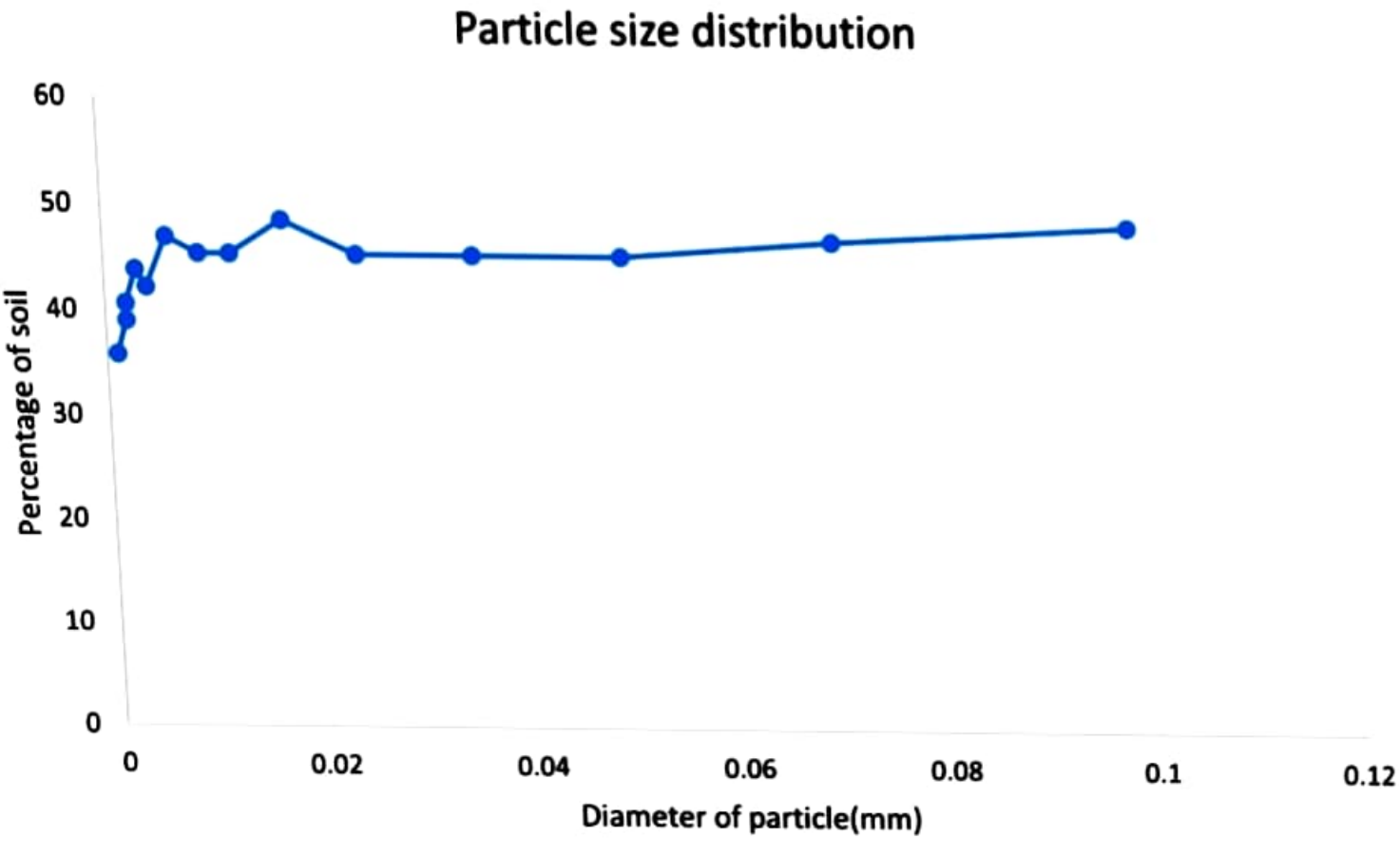
Result

→ Liquid limit = 25.06%

Plastic limit = 12.04%

Flow index = 3.59

Plasticity index = 13.02%.


Particle size distribution
60
50
40
30
20
10
0
Percentage of soil
0
0.02
0.04
0.06
0.08
0.1
0.12
Diameter of particle(mm)

Elapsed time t (min)	Temperature (°C)	Observed Hydrometer reading, R	Correction for meniscus Rm = R + cm	Temperature Correction C_t	Combined Correction C = C_m + C_t + Cd	Corrected Hydrometer Reading R_h = R + C	Effective depth HR	Particle Diameter D (mm)	Percentage fines P(%)
0	31.5°c	15	15.5	+3.8	+1.3	16.3	13.08	-	
0.25	31.5°c	15.0	15.5	+3.8	+1.3	16.3	13.08	0.097	48.6
0.5	31.5°c	14.5	15.0	+3.8	+1.3	15.8	13.28	0.069	47.01
1	31.5°c	14	14.5	+3.8	+1.3	15.3	13.48	0.049	45.42
2	31.5°c	14	14.5	+3.8	+1.3	15.3	13.48	0.035	45.42
4	31.5°c	14	14.5	+3.8	+1.3	15.3	13.48	0.024	45.42
8	31.8°c	15	15.5	+3.8	+1.3	16.3	13.08	0.017	48.6
16	31.1°c	14	14.5	+3.8	+1.3	15.3	13.48	0.012	45.42
30	31.6°c	14	14.5	+3.8	+1.3	15.3	13.48	0.009	45.42
60	31.5°c	14.5	15	+3.8	+1.3	15.8	13.28	0.006	47.01
120	30.8°c	13	13.5	+3.8	+1.3	14.3	13.88	0.004	42.25
240	29.8°c	13.5	14.0	+3.8	+1.3	14.8	13.68	0.003	43.84
480	28.5°c	12.5	13.0	+3.8	+1.3	13.8	13.88	0.002	40.66
900	30.2°c	12	12.5	+3.8	+1.3	13.3	14.28	0.002	39.07
1440	31.3°c	11	11.5	+3.8	+1.3	12.3	14.68	0.001	35.87