Cardiovascular disease IAmA draft answers - 12.11.19

https://www.reddit.com/r/IAmA/comments/dvbd5i/iama_cardiovascular_disease_researcher_exploring/

Some suggested questions to consider:

ANSWER:

POSTED XX:XX

Forgiii
1 point·**2 minutes ago**

Question from me as a paramedic: For us (at least in Germany) it's load and go. So basically save the patient from dying right away (checking pulse, blood pressure and treating them if the patient is dying from it) and get him to the hospital as fast as possible without moving him too much or not at all. Now is my chance to ask an expert on something I've always wanted to know. With heart injurys like heart attacks. Are there any special things me as an paramedic could do to further increase the chance of survival which we don't learn while becoming a paramedic Question from me as a normal guy concerned about the health of animals: How do you test this kind of stuff on animals. Is it cruel to the animals? Were there any deaths?

ANSWER: Hi Forgiii, thanks for your question. Paramedics are a vital first line with heart attacks, and what you do has been designed to give people the best chance until they can get to the catheter lab in the hospital. Any new things for you to do will have been thoroughly tested before going into wide use.

For animals, we anesthetize them and tie off part of a blood vessel in the heart to mimic a heart attack. Sometimes they stay under anesthetic to test acute treatments and are humanely killed at the end. Sometimes they are allowed to come round,and they will be treated with painkillers to prevent any discomfort. This is where we test longer term treatments, and we use echo and MRI to see if they are working. If they start to show any signs of heart failure they are humanely killed.

POSTED 15:54

Forgiii
1 point·**just now**
So you are saying that in every case the animal dies at the end because you manipulated the heart? I find that very cruel thou it's necessary to advance in medicine.

ANSWER: The law in Europe is very much about preventing suffering to the animal, quite rightly. Our biggest effort is to observe the animal very closely to make sure we pick up the very first signs that it might be in pain or distress. If we can't relieve that by painkillers or anything

else, we must end its suffering as quickly and painlessly as possible. We are not even allowed to keep animals into old age, in case they develop distressing ailments.

POSTED 16:30

poopellar
2 points·**3 minutes ago**
What kind of animals are these tests done on?
ANSWER: Mostly mice and rats, for the basic discovery experiments to find out new treatments. When they get close to being ready for people, they must be tested on larger animals such as pigs. We also use rabbits, because they may be enough like human in some heart aspects to be able to replace pig experiments.

Further information about our animal research and welfare at Imperial can be found here: https://www.imperial.ac.uk/research-and-innovation/about-imperial-research/research-integrity/animal-research/

POSTED 16:31

PTguy777
1 point·**2 minutes ago**
I was told in the medical program that I attended, that during heart failure the myocardium of the ipsilateral ventricle hypertrophies and becomes somewhat thicker with no subsequent positive inotropic effect. Could you explain why that is the case, since ordinarily the hypertrophied muscle would create an increased contraction force?

ANSWER: Hypertrophy causes an initial increase in force but changes in the cardiac muscle cell then make the force drop again, below the initial value. So even an increase in muscle mass does not increase the force of the heart at that point.

POSTED 15:55

SpecterTheGamer
1 point·9 minutes ago
Can you confirm that the number one cause of deaths by heart failures are because your heart suddenly stops working?
ANSWER: Hi Specter. You are talking about sudden cardiac death I think, when a massive disturbance of the heart rhythm stops blood from being ejected by the heart. Sudden cardiac death happens in apparently normal people, but not often. It also happens as part of a heart attack death. For heart failure death, about half the death is from this and half from a gradual failure of the heart to pump.

POSTED 16:02

agile_giraffes
1 point·**4 minutes ago**

Hello! I am looking into heart failure pathways and wanted to ask:

1. **what is the most common severity class at the time of diagnosis?**
2. **what would you like to see improved in the care pathway?**

ANSWER: I am a scientist not a clinician I'm not the best person so these questions.

POSTED 16:04

LOOKFURTHERLEFT
1 point·6 minutes ago·***edited 3 minutes ago***
I am taking loperamide for an opiate addiction because i can't find treatment.

I've been having edema in my chest muscles, but nowhere else. Is this a sign? The skin doesn't push in, but the muscle itself does. No fatigue or anything else.

ANSWER: This is best answered by a clinician

POSTED XX:XX

llyffant24
1 point·just now
Hi Sian. Have you always been interested in researching heart failure and cardiomyocytes and why did you decide to specialise in this area?

ANSWER: My father-in-law died of heart failure just before I went to University. I could see there was a problem that needed solving. And I had a project at uni which helped me explore this topic, so I was hooked.

POSTED 16:09

unhappygounlucky
1 point·just now
In your professional opinion is it true that if you tell a heart, an achy breaky heart, something that it might not understand the achy breaky heart might blow up and kill this man?

ANSWER: Absolutely! There is a condition called "Broken Heart Syndrome" and it can be caused by grief, excitement and sometimes by watching football! It can cause sudden cardiac death (which I've mentioned),heart rupture or another condition called Takotsubo syndrome, acute heart failure, which is often reversible.

POSTED 16:11

Coolwhip76

1 point·1 minute ago

This might be similar to what you studied or know, but my father has drop dead syndrome and he has his heart enlarged. He's got a defibrillator in right now and every couple years he gets the batteries change or something along those lines. Surprisingly he has lived past the limit that doctors set on him by almost half. He currently needs to take many many pills in the morning and struggles to exercise but does a lot of work with his hand and in the shop to keep active. Some of his Doctors say it's a miracle that he is still alive and I believe they're right. But my real question would be what would signs be that his health is declining even though he's already in such a (Bad state) as it is?

ANSWER: If he finds it harder to exercise, or he is breathless, or he has swelling of the legs. I'm sure his doctors will be looking out for these or any other symptoms though.

POSTED 16:14

GoodnightFairLady

1 point·just now

I know that the common atrial tachycardias you see in heart failure are due to overstretching of the myocardium, but what is actually happening at a cellular level that leads to the increased action potentials and/or ectopic pathways causing the tachycardias?

ANSWER: There are changes within the cardiac muscle cell itself, which modifies its electrical activity. The action potentials can become longer or the cells can become overloaded with calcium. But also there is a change in the muscle composition, as cells which die are replaced by scar. Then the electrical pathways across the atria get disrupted.

POSTED 16:21

Mungo_Clump

1 point·5 minutes ago

> **Deterioration of myocyte function during the development of heart failure is a process that is distinct from the original injury to the heart and may be the result of the body's attempt to produce maximum work from a damaged muscle.**

So could this be a case of the body not always knowing what's best? So if you found a way to block this reaction might modern medicine be able to provide a better post-injury treatment that doesn't lead to this type of heart failure?

Also. With all you know about hearts, what lifestyle changes to you enforce, or are you as daft as the rest of us?

ANSWER: Hi Mungo. The problem is that the systems that get activated when the body senses a loss of power in the heart are from ancient evolutionary times. Then, we were more likely to die of injury or accident. So the body is thinking you have to run away from a mammoth or have been bitten by a saber tooth tiger. They are for emergency, and are damaging if they go on too long.

In fact, the drugs we use now for heart failure are almost all to block the body's response

And yes I am as daft, if not dafter. But even I don't smoke.

POSTED 16:23

elpinguinosensual
1 point·2 minutes ago
What insight can your research offer front-line healthcare professionals like doctors and nurses? Any advice on patient education?

ANSWER: Last week I spoke at a meeting which explained the new therapies coming through now, like gene and cell therapy, to a wide range of healthcare professionals. These are going to be more and more important, and they have very different properties and potential to normal drugs.

POSTED 16:42

equinoxdark
1 point·1 minute ago
Sorry for not reading everything you have written in the details box and sorry for asking a normie question but: exactly how much exercise should an average person do to make sure he/she have no heart problems in life?

ANSWER: Its difficult to make one rule for everyone, but definitely more is better - until you get to extreme athletes when there is some doubt. Even a little is better than none.

POSTED 16:43

lyreflyn
2 points·36 minutes ago
Hi there, I'm currently an undergraduate student in the states, but one of my higher-level courses is in cardiovascular biology and this was one of our recent topics! I know a lot of research is being done on cell reprogramming, but I was wondering if any viable research has been done on cardiac fibrosis? As in, is it worthwhile looking into preventative treatments for fibrosis after an MI? Thanks for your time!

ANSWER: Yes there is a very active research field for this. But we need some scar formation after MI to prevent cardiac rupture. It’s getting the balance right between this and blocking the widespread fibrosis that causes stiffening and arrhythmia that is tricky.

POSTED 16:44

from EMarkDDS via /r/IAmA sent 2 minutes ago

As a dentist, the standard of care (a wonderfully ambiguous and ever-changing barometer) is to wait 6 months after a heart attack before treating those patients. Can you offer any guidance on which individuals are most at risk? What the line is between someone you'd be comfortable treating vs someone who should delay any elective treatment for a certain amount of time?

ANSWER: I'm not clear why this should be – can you tell me the reasons given for waiting this long?

POSTED 16:47

from blacksheep431 via /r/IAmA sent 9 minutes ago

What are your clinical sources of information if you have questions that your research cannot help decipher? Are there reputed journals or research websites you trust more than others? Thank for your time in breaking down HF!

ANSWER: Definitely the journals help, but I am very lucky to work in the National Heart and Lung Institute, where I have many fantastic clinicians working alongside me. It's important for biomedical scientists to keep up to date with the reality of patient treatment from active clinicians.

POSTED 16:51

from Trinilos via /r/IAmA sent a minute ago

In medical school we're taught 2 things that didn't make intuitive sense to me:

1. **Digoxin increases contractility and improves symptoms but does nothing to improve mortality. I'm presuming that mortality is associated with hypoperfusion, so why does a drug which improves cardiac output not decrease mortality?**
2. **AICDs are considered in heart failure patients so long as their EF > 25% or so. Why is this lower limit in place? It seems they'd still benefit from it, so my**

guess is this is a cost-benefit issue where patients aren't expected to live long enough for the surgery to be worth the risks associated with surgery?

ANSWER: Digoxin, like other drugs which increase contractility, can also increase the chance of getting arrhythmias and so are risky. Some even made mortality worse in clinical trials (so are not used of course).

I can't answer the second point I'm afraid.

POSTED 16:56

from sadlyecstatic via /r/IAmA sent 9 minutes ago

Is there a way to restore cardiac muscle? An artificial replacement? In the future could this be a better solution than a heart transplant?

ANSWER: The mechanical partial artificial hearts are doing quite well, and are supporting people for years as they wait for a transplant. They have the problem that they must be driven by an external battery, and the wire through the skin can cause infection.

We are working on engineered heart tissue (see this BBC article), which is made from pluripotent stem cells which we turn into cardiac muscle cells. The pluripotent stem cells can be reprogrammed from ordinary skin or blood cells, so we can effectively turn your skin into matching heart tissue. Theoretically, we could implant this in your heart without it being rejected. But there are lots of hurdles to go yet to make it safe and deliver it effectively for patients.

POSTED 17:00

from TurboKid1997 via /r/IAmA sent just now

What role does possibly "replenishing" the mitochondria have in repairing the heart after a heart attack? Are you involved in research into this? I have read about a case study where the heart was injected with the patients own mitochondria from muscle and it aided in the recovery of the muscle.

ANSWER: It's very clear that the mitochondria are defective after a heart attack, so will be producing less energy, and that in turn leads to a poor prognosis. I'm not directly involved in this though, and have not heard of the study you mention.

POSTED 17:05

from Daguvry via /r/IAmA sent just now

Can you elaborate on meth heart? What exactly destroys the right heart with methamphetamine use? Is it the meth itself (chemically) or how the meth effects heart function (physiological)?

I'm a Respiratory student and almost done. Any advice on anything for my career?

ANSWER: It's most likely the action of this drug to stimulate neurotransmitters such as catecholamines. Even the natural catecholamine heart stimulants, like adrenaline, act through receptors on the heart cell surface and can cause calcium overload and, if prolonged, cell death.

The National Heart and Lung institute is always looking for good young Respiratory scientists.

POSTED 17:10

DENTIST FOLLOW-UP

from EMarkDDS via /r/IAmA sent 5 minutes ago
Show Parent

For heart attack and stroke victims, the thinking is that the chance of a second incident is greatest in the first 6 months, so only emergencies should be treated. Our problem is, for example, my father's case; he had some chest pain and sweating, got a quad bypass, but according to the doctors suffered no heart damage. Can we treat him right away since technically he didn't have a heart attack?

ANSWER: I wouldn't really want to advise on your particular case, but I see the reason now.

POSTED 17:08

Follow up to "Hello agile_giraffes. Thanks for your question. I am a scientist not a clinician, so I'm afraid I'm not the best person so these questions sorry."

dronz3r
1 point·**5 minutes ago**

What's the difference? I was under the impression that all the medical professionals who went to medical school are doctors and can treat patients.

ANSWER: I did a degree in Pharmacology and then a PhD, so I'm a scientist. I work alongside clinicians but am mainly laboratory based.

POSTED 17:19

from simonbleu via /r/IAmA sent 3 minutes ago

What happens to the cardiac muscle during heart failure?

ANSWER: Heart failure starts with damage to the heart (e.g. from a heart attack, or chemotherapy drugs, or valve disease) or some loss of power in the muscle e.g. from a hidden genetic defect.

The body senses the loss of power and tries to stimulate the heart with adrenaline (and noradrenaline), by changing the shape of the heart, and by loading of water.

This causes further damage over the course of months and years. Eventually, either the heart gets weaker and heart fails completely or there is a sudden massive arrhythmia which causes rapid death. Drugs now are getting better at preventing further damage.

POSTED 17:26

from ocean_wavez via /r/IAmA sent a minute ago

Hi! I'm a nursing student in my last year. What are some signs of heart failure that are commonly missed? What can I do as a nurse to best care for those with heart failure?

ANSWER: Breathlessness can be confused with other diseases, like chronic obstructive pulmonary disease (COPD). In fact, because things like heart failure, COPD, kidney failure and dementia all tend to cluster together in older people, it can be difficult to pinpoint the primary disease. This is the current challenge of multimorbidity.

POSTED 17:30

from NealR2000 via /r/IAmA sent a minute ago

My brother unexpectedly passed away at 51 and the cause of death was Arrhythmogenic right venticular cardiomyopathy with predominat left ventricular involvement.

Can you please give me a layman's understanding of what happened?

ANSWER: I'm so sorry to hear this. Cardiomyopathy means a disease of the muscle itself, which seems to have started in the right ventricle (which sends blood to the lungs) and then involved the left ventricle (the main one that supplies the body).

Arrhythmic means that there were disturbances of rhythm of beating in the right ventricle. I can't tell from this whether it was the rhythm disturbance which caused his death in the end.

POSTED 17:38

Jayelvee23
1 point·32 minutes ago
Can having chemotherapy 20+ years ago lead to CHF? Or is this coincidental?

ANSWER: It could be part of it. There is a clear link between the drugs used to treat cancer and possible damage to the heart. As people are living longer after cancer treatment this is being seen more.

Cardiologists are teaming up with cancer specialists to prevent this happening, and to treat cancer patients when it does happen. Us scientists are trying to understand why the damage happens and design better cancer drugs.

POSTED 17:43

coldonewiththeboys
39 points·17 hours ago
How detrimental is the occasional use of cocaine for the heart of a healthy adult?

ANSWER: I once read a statistic which said that approximately one third of heart attacks in a London A&E on a Saturday night were cocaine-related. It activates the same system as adrenaline to cause arrythmias.

from Fire_Woman via /r/IAmA sent an hour ago

How significantly does alcohol affect the heart muscle? Is drinking ok?

ANSWER: There has always been evidence that light drinkers have less heart disease than teetotallers. The amount considered “light” has dropped a lot over the years. However, the latest research from Imperial shows that people with hidden genetic mutations in their heart can be more susceptible to the damaging effects of even moderate levels of alcohol

POSTED 10:40 (13 Nov)

Ware JS, Amor-Salamanca A, Tayal U, Govind R, Serrano I, Salazar-Mendiguchía J, García-Pinilla JM, Pascual-Figal DA, Nuñez J, Guzzo-Merello G, Gonzalez-Vioque E, Bardaji A, Manito N, López-Garrido MA, Padron-Barthe L, Edwards E, Whiffin N, Walsh R, Buchan RJ, Midwinter W, Wilk A, Prasad S, Pantazis A, Baski J, O'Regan DP, Alonso-Pulpon L, Cook SA, Lara-Pezzi E, Barton PJ, Garcia-Pavia P. Genetic Etiology for Alcohol-Induced Cardiac Toxicity. J Am Coll Cardiol. 2018 May 22;71(20):2293-2302.

POSTED 10:40 (13 Nov)

from theresalalala via /r/IAmA sent 3 hours ago

Hi Sian! I currently work for a cardiologist right now. We have pharm reps come often, and one of the newest drugs they have been talking about is one that they say is an rare / under diagnosed form of heart failure caused by amyloidosis. Do you think that Amyloidosis is actually under diagnosed? Also, what do you see the most pressing future study directions of heart failure?

ANSWER: Not sure how to estimate whether amyloidosis is underdiagnosed, but I suspect it depends whether you go to an expert centre with great imaging or a general hospital. One pressing issue at the moment is “heart failure with preserved ejection fraction”, which is where the stiffness of the heart is increased and it doesn’t relax properly. We are just finding out that the drugs developed for heart failure with reduced ejection fraction, where the heart is contracting poorly, don’t work on this other group.

POSTED 10:40 (13 Nov)

Additional text for blurb:

UPDATE [11AM ET / 4PM GMT]: And we’re LIVE!

Here’s proof that we’re here in person to answer your questions: TWITTER PIC

UPDATE [1PM ET / 6PM BST]: Thanks very much for your great questions everyone. I’m heading off for now but will be checking back in tomorrow, so please do submit any more questions you may have.

And a big thanks to r/IAmA for hosting this AMA!