**Presentation of Prof Arne Burkhardt – English translation**

Link for original conference, 18th Sept 2022 : <https://youtu.be/jLJXL3YlHKE>

**Subtitled video - Part 1**: <https://open.lbry.com/@LongXXvids:c/Prof-Arne-Burkhardt---2nd-Vax-Injury-Conference---Part-1:1>

**Subtitled video - Part 2**: <https://open.lbry.com/@LongXXvids:c/Prof-Burkhardt---Pathology-Findings--Sep-18-2022---Part-Two:c>

**Short video extracts**

Crystalline cholesterol: <https://open.lbry.com/@shortXXvids:e/Burkhardt---Evidence-of-crystalline-cholesterol-in-body:4>

Bblood clots: <https://open.lbry.com/@shortXXvids:e/Prof-Burkhardt-Mystery-Clots:e>

Aautoimmune diseases: <https://open.lbry.com/@shortXXvids:e/Prof.-Burkhardt---Vax-induced-autoimmune-diseases-observed:3>

Brain damage: <https://open.lbry.com/@shortXXvids:e/Prof.-Burkhardt---Vax-Induced-Brain-Damage:1>

Sudden Adult Death Syndrome: <https://open.lbry.com/@shortXXvids:e/Prof.-Burkhardt-short-extract-SADS:1>e

Aortic dissection: <https://open.lbry.com/@shortXXvids:e/Prof-Burkhardt---Dissection-of-the-Aorta:8>

**FULL TEXT OF TRANSLATION**

Updated 26.11.2022 with input from Prof. Burkhardt

Notes: text in *blue italicised typeface* provides an explanation of medical terms used which

may be unfamiliar to the layperson.

**Introduction by Dr Wolfgang Ruck:**

**00:06** I don't really need to introduce our next speaker, at least not here in the hall. We all know Professor Arne Burkhardt as the pathologist who, together with his colleague Walter Lang, also greets you warmly.

The two of them started the ball rolling with their investigations in Germany and worldwide. What does "started the ball rolling" mean? Professors Burkhardt and Lang were the first scientists to examine tissue samples from deceased people who were suspected of having died of the so-called vaccine. And, if I’m informed correctly, the first samples already showed that they were on the right track.

With the immune-histological differential diagnostic evidence of ths spike protein and its nucleocapsid *[a viral structure unit composed of nucleic acids and attached proteins]*

found at the beginning of the year by Doctor Mörz, the suspicion was confirmed and became a frightening certainty.

I’m very pleased that you are here and can tell us about the latest results of the pathology team.

**Prof Burkhardt:**

**01:30** Yes, I welcome you very warmly and I’m happy to be speaking here for the second time. Thank you for this invitation.

I’ve been working a bit hectically in the last few weeks to bring you something new because one knows from earlier times lectures at congresses where yellowed diagnosis slides were shown again and again. So I want to prove to you that this was not a selection of individual findings but that we really had a broad base of findings at our disposal – that is the Reutlingen study.

The participants have already been named, excepting some who do not want to be named. It actually began quite well. You certainly know the basics, but very briefly, in March of last year, an increasing number of relatives turned to pathologists suspecting that deaths might not be natural but related to vaccination. Most pathologists declined these requests and so they came to me for a second opinion and this is quite common in pathology. In autopsy cases it is rather seldom but in biopsy cases remains the rule. So, I said I'll have a look.

And then it was really as you just said. After the first five cases it became clear to me that there is a very complex and disturbing histological picture *[histology = The study of tissues and cells under a microscope ]* to find. The autopsies were done in pathology and in forensic medicine in about a fifty-fifty ratio. In forensic medicine there’s almost never any histology and in pathology it varies, but also partly not very much representative histology.

We then received the fixed residual material and were able to prepare sections to assess, and in addition to the autopsy cases we’ve also received in the meantime fifteen biopsy cases some of which I will spend some time on. And that will be of great importance in future because in a total of three runs, Mr Lang and I have found certain patterns which can actually be attributed to the vaccination, first of all the vascular damage that we have already heard about.

**04:15** It was not only dead, but also living people who were examined by us and the methods are outlined here [on the slide]. So, a quite normal histology as I have been doing for over forty years. Naturally, special staining, immunohistochemistry — services for this have already been mentioned which Dr. Mörz has contributed significantly to. And then we have various advanced chemical and physical methods so we work together with one or more chemists and a physical chemist. The particular problem here was of course — and therefore I do not blame the colleagues who have overlooked certain things — it is just that we have a toxicological problem before us and not a simple medical problem.

Toxicological problems actually belong to forensic medicine. Only the examination of the stomach or the … [corrects himself] … body fluids … But here there is a poison at work that is produced by the body itself, which means that one must actually look for this poison in the tissues and this was exactly our aim. And one must of course exclude natural infection but this is anyway clear.

I have Dr. Mörz to thank for this image. The first thing you can see here is that we have actually proved that the spike protein is produced in the muscle where it is injected. The dogma at the time was that it was only produced there, and the decisive thing is that we were able to prove that this can occur in almost all organs and cells under certain circumstances, and especially in the endothelium [*inner lining of the blood vessels*]. And in my prior presentation we’ve essentially shown individual findings from the [various] organs and here I want to put together what we’ve recognised as the so to speak overarching pattern.

**06:50** In the first place we have been able to detect the spike protein in almost all organs and tissues in those cases that had the corresponding indications. Then the continuous finding and actually the common denominator of all these versions is endothelial inflammation — so called “endothelialitis”, a terrible term which unfortunately is internationally common. A destruction and inflammation of the endothelium has already been discussed but specifically, what has occupied us in the last weeks are what we initially classified as unidentified foreign bodies or deposits.

I must say that these so-called foreign bodies were actually overlooked in the first round of examinations I did.

Why did we overlook them? Because the pathologist knew the so-called formalin pigment. This is a recognised artefact so the pathologist automatically dismissed it, but at some point, we said that can't be true, it is so selectively distributed. And then the same applied to what we classified as ‘unidentified protein’ deposited in [blood] vessels.

Then also completely new: the problem of the so-called clots, so clots that don’t have thrombotic clumping which are seen in the blood of both living and dead people. We just got a sample from a living patient.

The actual foreign bodies coming from contamination originating from the vaccine … we only have two cases of this and for us, when considering the adverse effects or cause of death, this is at present playing a lesser role.

Here next, the conclusive proof that the protein, which is shown here in the fatty tissue, that’s these vacuoles, which was brought in here so really quite selectively here a capillary is coloured, so the endothelium is producing active spike protein here.

**09:29** Here is a bifurcation, and here it is lying outside the place of the section, and that is the proof, and we were able to show not only from post mortem cases but also with biopsies.

We were initially strongly attacked after the first conference. It was said that this endothelial damage is all autolysis *[degradation caused by the cell’s own enzymes]*. But that is all screwy because this is from a living woman who had severe circulatory problems on the thighs and legs and where you see the endothelium is actually even detaching here and is swollen. Small [blood] vessels do not normally look like this – and here is the spike protein – you can see the clear marking of the endothelium and here practically a closure / occlusion and ultimately an obliterative vasculitis *[Vasculitis is a group of disorders that destroy blood vessels by inflammation. Both arteries and veins are affected.]* And we have to imagine this and explain why it is so worrying.

This illustrates the inside of a blood vessel with blood cells and the lining of the endothelium – these long drawn out cells. And when these cells are destroyed by the insertion of the spike protein then this naturally has the result that thrombocytes attach themselves here and thrombi [blood clots] can arise.

But in my view, it’s also very dangerous that the spike protein in this way doesn’t just remain in the blood vessel but actually gets through the basal membrane and into the elastic lamellae which then plays a big role in vascular damage.

**11:24** And I will now start with the first problem we are still, so to speak, trying to crack. In the first conference I had shown this case. It is in the lungs but obviously not in the alveoli, not in the air sacks, but this large foreign body cell. At the time I had just read an article about precious metal particles being present in the vaccine so I was somehow fascinated.

And it then also occurred to me in retrospect that it’s a phenomena that we all know well – it is these so called cholesterol needles. And it was unclear how they came to be in the interstitium *[a supporting or connective tissue or material that lies between the principal cells of an organ, or between structures in the body].*

And it was then said that something must have been inhaled by the woman. But first of all, it is noticeable here that there is then a birefringence *[double refraction of light]* in the marginal area and we also find this material in the spleen. I will show that later.

And we find this foreign body material, which we had initially thought was formalin pigment, is here in massive deposits in the pancreas where it’s most frequently seen and afterwards in the spleen and partly also the heart and as good as never in the brain. And you can see that these are vacuoles with a mild inflammatory reaction here in the surrounding area.

And these vacuoles — they are naturally not empty as they appear here. So while alive, there was a lipoprotein inside, a type of fatty material which has been dissolved out through

the treatment with alcohol, xylene and the like. But something was in this foreign body material inside the vacuoles that got left over and was resistant to the embedding.

**13:37** So, I’ll show various pictures now. And none of this is compatible with formalin pigment.

This is a non-dyed sample. And here again these partly predominantly rod-shaped structures. And interestingly, quite often in the immediate neighbourhood these swellings. The eosinophilic deposits in the blood vessel walls may be related to this. And also for this material which remains in the vacuoles, we see a birefringence, not always, but often.

And we followed this up using so-called Raman spectroscopy, which is very laborious … so we started slowly, because we first have to set up all the conditions as this method is mostly used for material investigations and not in the biological field. And we naturally had to use non-dyed sections for this work, and above all, the foreign bodies we wanted to investigate, which for example you can see here, we needed to position them [carefully], because if you are using the 100 or 1000 times magnification then you suddenly don’t know where you are. So, we have to establish where we are and direct the corresponding beam [of the spectroscope].

So here are the first spectra. And this is the important one here – this peak is paraffin, and these other peaks are difficult to identify. This will be measured and compared. We are still working on this but the physical chemist who did the analysis is fairly certain that this is cholesterol.

**15:59** Cholesterol normally circulates in the blood as lipoprotein and not as pure crystalline cholesterol. And as such, it is found in gallstones. And we have the supposition

that the cholesterol crystals we see are derived from the lipoproteins – they appear very angular.

These here are from textbooks, but they could be just pure cholesterol crystals which don’t normally occur in the body but they’ve remained as a residue after the lipoprotein was dissolved away.

And here now, the pattern of damage to the large vessels. You see here the interior. This has a significance because we have investigated where the cholesterol can come from. The amounts that we have found are not compatible with a source from the vaccine itself. So it could be that there is an influence on the metabolism of cholesterol. But where is the cholesterol? It is in the atheromatous plaques of the blood vessels. *[ atheromatous plaque, is an abnormal and reversible accumulation of material in the inner layer of an arterial wall.]*

And next here, a diagram of a large blood vessel, in this case the aorta. You see here that the spike attacks in two places. In the first, it naturally damages the endothelium of the large blood vessel and then the arterial intima breaks open, in which case the cholesterol which is in the atheromatous plaques can just enter the blood. This is something rarely observed in pathology but which can lead to atheromatous embolism.

And then comes this second damage mechanism via the vasa vasorum *[small blood vessels that supply or drain the walls of the larger arteries and veins and connect with a branch of the same vessel or a neighboring vessel]*, which has already been mentioned earlier. And from here the supply takes place from the outside … and here from the inside … and the media *[the middle coat of the wall of a blood or lymph vessel consisting chiefly of circular muscle fibers.]* stays here and it is susceptible to necrosis from a toxin or something similar in the blood.

**18:22** And this is our current concept – this is a diagram from a 1980 medical textbook ‘Pathogenese’ by H.Cottier. Here you see these needles and the covering of the endothelium. These are the famous atheromatous plaques and when they break open due to the spike protein causing them damage, they are washed away into the blood. That is our concept. The chemist has found pure cholesterol, so similar to salt or sugar. We will now embed this, cut it, and see how a section of this crystalline material looks.

And I spoke with a chemist in Vienna who said that cholesterol crystals can be tremendously polymorphic, which means that there is no fixed pattern [in their appearance] It could perfectly well be these rod-like structures. And in our cuts, we actually have an incidence of such an obviously broken intima membrane *[This is the thinnest layer of the vascular wall consisting of a single sheet of endothelial cells resting on a basement membrane and a thin subendothelial extracellular matrix (ECM) composed of collagen and elastin.]* which has been bleeding. At least this is our working hypothesis. I want to say clearly, this is no absolute evidence but the evidence is naturally the prepared cuts [tissue sections].

And now the second unidentified discovery or material. That is these strange eosinophilic deposits *[stained preferentially by Eosin]* we observed in blood vessel walls which we also initially did not consider, but of course there are all sorts of things – but it was striking that we found this yet again.

**20:17** And then we also talked about the unidentified protein material, and then came the epiphany when a Swedish science group just proved that the spike protein is identical over long distances with another protein which is called amyloid. That means starch like, it’s an eosinophilic material having an affinity to Congo red stain and gives a birefringence.

And there is a disease called amyloidosis, *[*A*myloidosis is a rare disease that occurs when a protein called amyloid builds up in organs.]*where the heart muscle, the lungs and the spleen are involved and above all — and that’s the worrying thing — the brain is affected. There are micro-aneurysms, [aneurysm= *a cerebrovascular disorder in which weakness in the wall of a cerebral artery or vein* causes a localized dilation or ballooning of the blood vessel*]* bleeding, and senile plaques relating to Alzheimer’s disease. [Amyloidosis] is a known disease that occurs in chronic inflammation for example in chronic bronchitis in predominantly elderly people.

And as I mentioned, this is known, and you can look here – this is a medical check sample – a positive heart muscle where you can see it here with the Congo red staining. And here now is the corresponding findings in the histological section. We find this picture disturbing in relation to these deposits, also in the vessels, so partly occluding. And in fact we were able to evidence these Congo red deposits, and with that detect an amyloidosis — or at least evidence an amyloidosis band of these deposits in the spleen artery [with] clear positivity of the inner band layer of the endothelium.

And here now is this thrombosis-like layer, no real thrombosis but rather just protein here. Also, inflamed cells are present. This, in my view is a very clear indication that there is really an amyloid-like material present. That is why we also speak of functional amyloidosis which means that it should have the same effects as the real disease. Whether it is chemically absolutely identical is another question.

**22:46** And here is the fatty tissue from a living female patient. You have material deposited here in the periphery and sporadically then also in the connective tissue fibers — positive fibers. Whether these really all contain amyloid [protein] we will also examine further using electron microscopy.

It is interesting that these crystalline deposits of ‘fatty materials’ are often enclosed by amyloid. So directly around this is amyloid and I had already shown it in the adjacent vessel that these deposits are also present.

Next, we now have two cases where in the lung we have these rather strange fiber structures which are also Congo red positive and partly interstitial, but also they partly go onto the alveoli. And this here shows just next to the Congo red colouration a birefringence. I have here a half-darkened image. I wanted to have seen absolutely the identical place. Here one quite easily sees a colouration in red. And here there is clearly a typical birefringence. It is a bit more variable and not so even as in the typical cases for amyloidosis. And the colour depends a bit on the optical system but it is supposed to be apple-green — with such comparisons it’s always a bit difficult but I think in any case it is a birefringence which colours with Congo red, so it is amyloid like and has the same biological significance.

**24:53** Now I come to the next problem we are dealing with. That is the [blood] clots. They are not thromboses and they were first seen in corpses, corpses that were of course stored in a refrigerator.

They appear stretched / elongated, and are somewhat yellowish-white and elastic. And so far no one has been able to find out exactly what they are. One thing is for sure — they must have been formed after death and during the cooling process because no one would have been able to live for long if all the [blood] vessels were filled [with this material]. So one dies already when the first vessel closes, which of course is also possible.

And it is now chemically proven that it is not a blood clot, which was already clear from the optical perspective. This was now a chemical analysis. *[reference to work of Mike Adams]*

This is now the woman already reported on by Dr. Mörz — a forty-year-old and former marathon runner who was very good on her feet and everything and had been vaccinated and then had these extreme circulatory disturbances in the legs and feet.

And this image I believe we have seen. Here, when you take her blood and centrifuge it, then, upon cooling it forms this clot, or whatever you want to call it down here. Underneath is the blood and an intermediate layer. This was offered to us for histological examination. I actually declined this and explained that blood dissolves away when you prepare a section for histology but we did however, on the wishes of the patient, carry out an examination. It looks like a whitish quite solid material which is a bit elastic and actually during the whole embedding process it is not dissolved which surprised me.

**27:11** Yes, and so it looks like this. A clot that has small outgrowths and you can think, or have the feeling that it grows, which means it’s a phenomena that is then called self-assembling proteins, that is, the proteins are in the blood, and when the blood is warm they are separate, and when it cools down, they form a pure protein mass. And [the clot is] practically cell free so there are no fibrocytes or fibroblasts *[ Fibroblast and fibrocyte are two stages of fiber-producing cells in the connective tissue. A fibroblast is the active form whereas the fibrocyte is the inactive form. This is the main difference between fibroblast and fibrocyte.]* that formed the tissue [structure] in it.

There are no erythrocytes *[red blood cells]* in there — so I found three but that is practically none. And the lymphocytes *[white blood cells]* are scattered. Why of all things the lymphocytes are baked in here so to speak and not the erythrocytes remains a mystery. By the way, such a fibrous tissue — what kind of fibres they are we will also check.

We have an offer from a Dutch university who wants to investigate this with a mass spectrometer and interestingly enough, there are such condensed structures in there that are not due to [sample] cutting artefacts. And that is the stain colouration. And this is at high magnification with the EvG-stain.

So if one really wanted to make an assumption, then these could actually be fragments of elastic fibres that have somehow detached themselves from the damaged vessel’s intima. But these are now, as I said, pure assumptions and without more we cannot go further.

**END of Part One (28:57)**

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**Part Two**

**00:05** And then we had another interesting finding from a lung. We saw in the [blood] vessels — but not attached to the walls — what we first classified as foreign bodies. They were not birefringent and, as said, not attached to the walls, which ruled out embolism and thrombus [formation] and we were able to detect spike proteins. We were also able to detect amyloids in it, and fibrin. And my idea is that this is only formed post-mortem during cooling, and it could have grown even further than that — it could have been the seed from which these clots were formed.

Yes, these are the general findings which I have newly shown. Now we come to the specific organs and the tissue lesions.

These small [blood] vessels are in the first place, with changes proceeding up to obliteration, and where we find in the walls, under certain circumstances, this spike protein-fibrin-amyloid material. It looks like that — the endothelium is detached and here are all the inflammatory lymphocytes we’ve already seen. So that is certainly not an autolytic effect but there is certainly an intravital effect. *[intravital= occurring within, or performed upon, an organism that is alive ]* And we can then actually — this is a periappendicitis but from another pathology institute — we can actually evidence the spike protein in this periappendicitis, in the damaged epithelium.

**02:10** Decisive here — these are the thrombocytes which are here picked out with CD 61 [stain] and they have attached, plaque-like onto the vessel walls and can then contribute to the thrombus formation.

Then, the large [blood] vessels — that is also very worrying because not in every autopsy are larger blood vessels looked at and the aorta is not regularly examined histologically and forensic pathologists practically never examine histology anyway. We have found a textural disturbance of the [aorta] wall — I had shown you the texture earlier in a diagram — practically in all cases to a small extent or up to dissection.

Dissection, perforation, at that time we had 40 cases, and 5 cases died of rupture of the aorta and these are findings predominantly from other pathologists — these thrombotic outpourings which was already partly talked about where we don’t know exactly what is going on.

And I’ll repeat this again — The inner half of the blood vessel is supplied here — from the intima, and here the outer part from the vasa vasorum. And here, so to say, is the Achilles heel of the [blood] supply. And exactly there, attack the infectious, toxic and other processes because that’s where the blood supply is the worst.

**04:05** And now I come to an historical medical picture, which I immediately thought of when I examined the first histological section. There is much more objection because for such dissections i.e. vessels folding apart, when has one seen that?

Well, yes, for example with Mesoaortis luetica *[a form of cardiovascular syphilis]* with HE staining and that is a historical slide preparation.

And that just shows a practically no longer existing finding. And I also remembered that in my early assistant days I once saw a case of lathyrism *[a neurotoxic disorder chiefly affecting people and domestic animals (such as cows and horses) that is characterized especially by irreversible spastic paralysis of the hind or lower limbs and that results from poisoning by an amino acid found in the seeds of some legumes (genus Lathyrus and especially L. sativus)]* dissection of the aorta resulting from a toxic agent from chickpeas, which occurs no more due to food control measures.

And at this opportunity, I would like to stress that we have been criticised for presenting findings for which we have no proof. Now here are the proofs preserved for more than a century. This preparation dates back to before the First World War and Mesoaortitis luetica is still around. And [these slides] can also be evaluated and everyone is invited to take a look at our preparations and see that the findings are practically identical. Here, infectious toxicity — a fissure formation. Here the intima, and here the adventitia, and here the inflammatory infiltrate, and a haemorrhage.

**05:50** So that’s what I was thinking of, and that has been confirmed rather dramatically in the cases which we have. I mean even the layman can see that here the blood would be flowing — here the surrounding tissue and here everything is simply broken and there it opens up and tears.

And there you see the inflammatory infiltrates, and here these fissure formations — clear proof that this concerns an event before death and that autolysis has not played a role.

And then also here, the inflammatory infiltrates and Dr. Mörz claimed to detect the spike protein, mind you specifically in the lesions.

And then of course the inflammation in the vasa vasorum. And here then, the disturbances and destruction of the elastic lamellae and as said, I ask myself — where are these fragments of the elastic lamellae? Might they get into the blood and form these clots? But as I said, this is pure speculation.

Yes, and this concerns not only the aorta but also other [blood] vessels and as I had already said, in practically all the larger vessels that we have examined. As I said, they were not taken from every case.

**07:09** We have been able to prove texture disturbances, not always in the sense of a dissection but somehow a bit of irregularity. And here is a coronary artery and you see this person had no significant arteriosclerosis because here the arterial wall is actually quite normal and then suddenly here is a focal destruction as one calls it — and this is exactly the same as the medial necrosis *[a lesion of the aorta in which the media (the middle coat of the artery) deteriorates, and, in association with arteriosclerosis and often hypertension, it may lead to a dissecting aneurysm.]* that we saw before in the aorta.

And here it is again with inflammatory infiltrates. And here, cystic medial necrosis as it is called. *[CMN is a disease of large arteries, especially the aorta, caused by collagen linking defects leading to deposition of basophilic ground substance in the media, creating cyst-like lesions that weaken the artery wall.]*

By the way, there is also a congenital form of medial necrosis aorta which occurs however without inflammatory infiltrates. That means it dissolves the media away in a similar manner but without inflammatory infiltrates.

And there, the coronary artery — the spike proteins nicely shown here in macrophages *[a large phagocytic cell found in stationary form in the tissues or as a mobile white blood cell, especially at sites of infection.]*

and myofibroblasts *[Myofibroblasts are a differentiated cell type essential for wound healing, participating in tissue remodelling following insult.]*

Then here, another coronary artery — we see these extended haemorrhages — inflammatory infiltrates again as evidence. Here, an immunohistochemical stain where the spike proteins have been detected. And here you can see that this is also infiltrated in the wall, and then not only the main artery [but also] the carotid artery, [and] the renal artery — you see fissure formation.

**08:46** Here, the main vein of the spleen with this dissection, and finally also, a brain artery with this dissection and scattered inflammatory infiltrates. Yes, so that’s a regular finding and for me it is associated with vaccination and thus it is conditional insofar as the spike obviously attacks the elastic [lamellae] or the cells that are responsible for it.

And it is now no wonder that suddenly a term has appeared that I never heard before in pathology for forty years — Sudden Adult Death Syndrome (SADS) — whereby — when I go through this it’s conditional, and also why the dissection of brain cells supplying the arteries is now suddenly taken as a topic in the most recent medical journal. So somehow, attention must have been drawn to this fact.

And for those who want to have more impressive images of it, here we have the aorta where the blood would have flowed. And it’s important in this context that we only have a slight arteriosclerosis here. So, there are no beds that appear to be somehow broken open or anything like that.

**10:09** We have these two layers here which was also shown earlier on a small preparation. Here you can see the outer wall, so to speak, and here the one in-between where the haemorrhage took place. And here again, it is very clear that there are dense inflammatory infiltrations and in these we can detect the spike proteins and this is neither compatible with an idiopathic media necrosis *[a lesion of the aorta in which the media (the middle coat of the artery) deteriorates, and, in association with arteriosclerosis and often hypertension, it may lead to a dissecting aneurysm.]* nor with syphilis in this case or similar inflammatory infectious event.

Yes, very briefly an addendum to what has already seen said in other conferences — Here are the main findings in the lymphatic organs — activation or depletion … then in the spleen the typical onion-skin type arteriolitis *[inflammation of the arterioles]* that we find in autoimmune diseases … wall defects, necroses … infarctions … then the unidentified fatty material that I’ve already mentioned … a malignant lymphoma which we examined.

That is the onion-skin arteriolitis where you see the [vessel] wall is broken up. You see eosinophilic-stained bands already here and actually we can then also evidence the amyloid deposits using Congo red staining and we can also find the spike proteins in these loosened wall vessels.

Then a lymphoma here, a pseudo-lymphoma with a considerable size where we have not yet seen a malignant lymphoma. But then here, a very rare B-cell lymphoma of the gastric mucosa. Here, the tumour cells are shown — they are really brown coloured, and here you see a dense inflammatory infiltrate, but not necessarily in the sense of a gastritis, but probably as a stromal reaction *[indication of an accumulation of inflammatory cells, fibroblasts and small vessels surrounding malignant cell nests of different tumors e.g., breast, colon, and pancreatic carcinoma]* so an immunological reaction to the tumour.

**12:18** And now the interesting finding — You see here, like a negative picture in the next case, the spike protein and you see the tumour itself is negative but predominantly the lymphocytes and macrophages are so clearly expressing the spike protein. About the connection, I can only speculate here but it cannot be a coincidence that something develops like this.

Incidentally, the patient has decided not to treat it now but to observe it because it is such an unusual case and in the clinic they also believe in a connection with the vaccination. And the hope is that it can perhaps disappear.

Then of course we also have the heart, lungs and the non-lymphatic organs.

About the heart I don’t need to say much. In the meantime the health officials now internationally recognise this as vaccine damage. There is no need to argue about it at all and in my eyes all doctors who declare this to be harmless are simply irresponsible.

And in the lungs we have this lymphocyte rich interstitial pneumonia.

Yes, the heart muscle as said [with] lymphocyte infiltration and destruction.

Then to distinguish it from a real infarct *[a small localized area of dead tissue resulting from failure of blood supply]*one wakes up to the fact that Chloroacetate-Esterase is expressed by the inflammatory cells and at the first moment one thinks of granulocytes *[a white blood cell with secretory granules in its cytoplasm, i.e. a neutrophil, basophil, or eosinophil]* and that this is typical of an infarct. But we could then prove that these are mast cells *[cells which play an important role in how the immune system responds to certain bacteria and parasites and they help control other types of immune responses. They contain chemicals such as histamine, heparin, cytokines, and growth factors.]* that spread here in the myocardium.

**14:01** And the image here is from Mr. Mörz. He has nicely shown that these mast cells actually degranulate in the myocardium, in fact just like small Molotov cocktails. So, they have everything that’s bad and contain everything that triggers inflammation and damages the tissues.

And also this preparation from Mr Mörz — the endothelium appears very specifically coloured. However, we are not quite clear if this expression of spike proteins in the heart muscle itself is to be assessed in the negative — well at least it does not look stained. Whether or not this plays a pathogenic role, I or we, if I have understood this correctly,

would like to leave open.

Yes, that is a lung with these very conspicuous knotty lymphocytic infiltrates which led us to use this term ‘lymphocytes amok’.

But we have another finding that has not yet been presented, and that is this lung. We now have two lungs — if we only had one I would have to say it could be a coincidence because we have these thread-like deposits in the interstitium *[a supporting or connective tissue or material that lies between the principal cells of an organ, or between structures in the body.]*

and in the alveolar lumina.

That is the one lung and this is the other one. So, similarly fixed and coloured — that is quite clear. And also this one here is coloured as amyloid with Congo red. Yes, and now this finding will be further examined with Raman spectroscopy to assess to what extent this is foreign material. One has the feeling that the amyloid actually only binds to existing fibrous structures because it is not so continuous as one would otherwise imagine.

**16:05** Then one more finding. It has already been actually spoken of as the favouring of inflammatory process in all organs. And here, a urologist called me and said he now sees lymphocytic infiltrates in the prostate gland. We’ve found them in the testes — I’ll come onto this soon but that is a new finding.

A dentist explained he suddenly sees exploding periapical granuloma *[a relatively common lesion or growth that develops around the tip of a tooth's root. It consists of a proliferating mass of granulation tissue (new tissue that forms on a wound) and bacteria that forms in response to dead tissue in the pulp chamber of the tooth.]* in his patients and sent this to me.As you see here, a partly granulomatous inflammation, here a small calcification, here scarring, and here we have the spike protein, nicely surrounding a [blood] vessel here but also elsewhere. So obviously a considerable inflammation promoting mechanism probably with an pre-existing apical granuloma that was small but now suddenly became explosive like the development of tumours which we’ve already heard about.

**17:12** Yes, for this we coined the term ‘lymphocyte amok’. Some colleagues disparagingly said such a term doesn’t exist. But at some point you have to coin a new catch-phrase. We understand under the “lymphocyte-amok”, a lymphocytic accumulation, partly with nodular formation and also germinal centre formation in non-lymphatic organs and tissues. And this inflammation in our view carries the danger that it can develop autoimmune diseases.

Here is the definition of the lesion — it is related to Wegner’s granulomatosis *[an uncommon disorder that causes inflammation of the blood vessels in your nose, sinuses, throat, lungs and kidneys. Formerly called Wegener's granulomatosis, this condition is one of a group of blood vessel disorders called vasculitis. It slows blood flow to some of your organs.]*  and further [conditions] like polyangiitis *[inflammation of multiple blood vessels or lymph vessels.]*  and other autoimmune diseases.

And now here are these lymphocyte aggregates, often around vessels — here in the lungs. So you don’t see that in a normal lung of course. You also find lymphocytes in the lungs but of course in the hilum area of the lymph nodes, *[The hilum is what connects your lungs to their supporting structures and where pulmonary vessels enter and exit your lungs.]* But this is not in the perihilar region *[the area surrounding the hilum of the lung]*.

And here, that is the perivascular lymphocytic infiltration.

And then also very disturbing, in the dura mater *[the tough outer layer of tissue that covers and protects the brain and spinal cord and is closest to the skull. The dura mater is one of the three layers that form the meninges.]* such lymphocytic infiltration.

And here is a second case. By the way that infiltration is a little less pronounced.

And here are the locations where we’ve found [infiltrates].

**18:56** So you can see for yourself — practically all non-lymphatic organs are affected here and also muscle tissue and fatty tissue can be affected here, also outside the injection site. And we have several cases [of autoimmune diseases] that developed after vaccination — so two Hashimoto thyroiditis *[Hashimoto's thyroiditis, also known as chronic lymphocytic thyroiditis and Hashimoto's disease, is an*[*autoimmune disease*](https://en.wikipedia.org/wiki/Autoimmune_disease)*in which the*[*thyroid gland*](https://en.wikipedia.org/wiki/Thyroid)*is gradually destroyed.]* and two Sjoegren’s syndrome *[ a disorder of your immune system identified by its two most common symptoms — dry eyes and a dry mouth ]* and a Lichen planus *[a condition that can cause swelling and irritation in the skin, hair, nails and mucous membranes. On the skin, lichen planus usually appears as purplish, itchy, flat bumps that develop over several weeks.]* — So real and known autoimmune diseases that have developed obviously only post vaccination, and which were at least not known before. It is of course possible, that they were already present subclinically but then through vaccination they have, so to speak in a figurative sense, received a booster.a

Yes, that’s not a very nice picture but that is the Lichen planus that we observed in a woman who, as she said, before vaccination had an absolutely clear skin and now can practically no longer go to the swimming pool because her skin is so disfigured.

These are no erythrocytes here but this is the basal region of the epithelium. Those are destroyed basal *cells [A small, round cell found in the lower part (or base) of the epidermis, the outer layer of the skin ]* and these are so-called hyaline bodies which one sees in Lichen planus — and of course here, the dense inflammatory infiltration — and then also, under the epithelium this vasculitis — so this inflammation. Here also, the swelling of the endothelium — I think it’s quite clear.

**20:28** And then we come to the brain — as practically the last organ we want to examine, which of course has a special significance. The transfection-associated-encephalitis was already presented to us and also published … then we have a lymphocytic infiltration … and focal destruction of intracerebral … and subarachnoidal blood vessels … with and without haemorrhage … with and without aneurysm … I already showed the dura mater infiltration … Dr. Mörz also observed this partial necrosis of the hypophyseal gland … and we have the amyloid *[a waxy translucent substance consisting primarily of protein that is deposited in some animal organs and tissues under abnormal conditions]* deposits.

And here, for those who are not so familiar with the anatomy — We have the skin, the skull, and just here and here and here, the lesions.

And I’ll now show you what is known about Haemorrhagia per diapedesis — *[the passage of blood cells through the intact walls of the capillaries, typically accompanying inflammation.]*

so, transmigratory haemorrhaging that could lead to these small point-forming lesions and possibly later even lead to larger bleeding.

But our findings are different in that we primarily have a haemorrhage in the vessel wall so we actually see an intravascular hematoma if you like. And I must say I am shocked to have found vascular changes in virtually every brain we examined, a fact that cannot be swept under the carpet.

**22:16** You can look here at the case numbers — these are now mainly other cases we have observed. Here a small capillary [with a] clearly swollen and damaged endothelium. Here a vessel complex with lymphocytes and haemorrhaging — small haemorrhaging which does not lead to death mind you. And here, lymphocyte infiltrates around a small blood vessel.

Here, at the same time, two vessels which are inflamed and altered. The smaller one shows brown deposits around the vessel — as said all different or mostly different cases — we then found partly iron or occasionally lipofuscine pigment, which must have originated from perished neural cells. This has to be examined in each case by special staining.

Then here, these small thrombi — micro thrombi. And here again hyalinization *[a process whereby tissue degenerates into a translucent glass-like substance]* of the vessel walls.

Then as Dr. Mörz has already shown, we have actually been able to evidence the spike protein here in some cases — and also quite specifically here in the endothelium.

And now we come to the micro-aneurysms illustrated here in a diagram from H. Cottier, my pathology teacher. It goes effectively up to bleeding, but the bleeding that we predominantly observe or often observe that does not necessarily lead to death are these intra-mural bleedings. And to make it clear that is the endothelium here, and these are the elastic lamellae — it is also clear that they have a continuous course. You have to imagine the pressure is very high there.

**24:14** We have the smooth muscle fibre there and, as said, these elastic lamellae have to be continuous throughout. And instead, we have pictures like this. These are the amyloid deposits — so these protein dissolving deposits in the loosened and somehow half-destroyed vessel walls. And we have then effectively also here shown in black, the elastic lamellae — and you see these are completely irregular and jumbled up and we’ve stained the amyloid in red.

And this is now a classic picture of a micro-aneurysm which can burst at any time. You can see these ripped and somehow completely irregularly arranged and partly destroyed elastic lamellae. Then you see here the amyloid deposits [stained with] Congo red and you see then here the interruption and here it has come to thin protrusions — a micro-aneurysm and that can burst at any time and lead to massive bleeding.

Here is a similar picture — the amyloid, then these elastic fibres all mixed up and black coloured, and that is actually the real colour, and what is it? Obviously, iron has accumulated on the elastic lamellae which then leads to these lesions. So this here is, so to speak, shortly before the mass haemorrhaging, and did not cause death.

**26:02** Yes, and a colleague wrote to me and said — Oh, that’s all calcification and I see that more often in the brain, but — and that blew both of our minds — we found similar findings in an artery of the thyroid gland that I’ve never observed in my whole life. So it must be vaccine associated.

So that was the slide showing the thyroid gland but now the really very disturbing finding — this plaque-like deposition of Congo red [stained] amyloid material as we find in neurodegenerative diseases which can result in bleeding.

**26:46** And that brings me to the end.

So that is the situation in which I found myself and in which we found ourselves after this after the first pathology conference. Everyone fell upon us and said — where is the evidence? I said the evidence is in the histological sections I’ve showed you which you can still look at after a hundred years.

Not a single pathologist, except of course the ones present here, accepted the invitation to take a look into the matter. From that moment on it was clear that we could not just give a second opinion but had to handle this as a scientific project.

We’ve made an effort — and quite an effort. In the beginning I was alone but now have colleagues who are involved in this. We are trying to make diagnoses while fighting with a lot of problems but I don’t want to go further into that here.

Yes, this cartoon was once meant to be amusing but it has become the bitter truth. I’ve had up to twenty calls from patients or relatives of deceased patients complaining bitterly that doctors don’t listen to them. Doctors don’t listen so that means now it is the pathologist’s turn. I have to take over this pastoral care, so to speak.

---------------- END (**28:26**) -------------

Disclaimer: We have taken every effort to provide a working translation which can be useful by both lay people and professionals in helping understand Prof Burkhardt’s findings. We take no responsibility for any remaining omissions / errors.