SUPERIOR COURT OF THE STATE OF CALIFORNIA

COUNTY OF SAN FRANCISCO

DEWAYNE JOHNSON,

Plaintiff,

vs. Case No. CGC-16-550128

MONSANTO COMPANY, et al.,

Defendants.

_____/

Proceedings held on Wednesday, July 18, 2018, Volume 12, Morning Session, before the Honorable Suzanne R. Bolanos, at 9:25 a.m.

REPORTED BY:

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Pages 2512 - 2625

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(None.)

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Morning Session

San Francisco, California

Department 504

Judge Suzanne Ramos Bolanos

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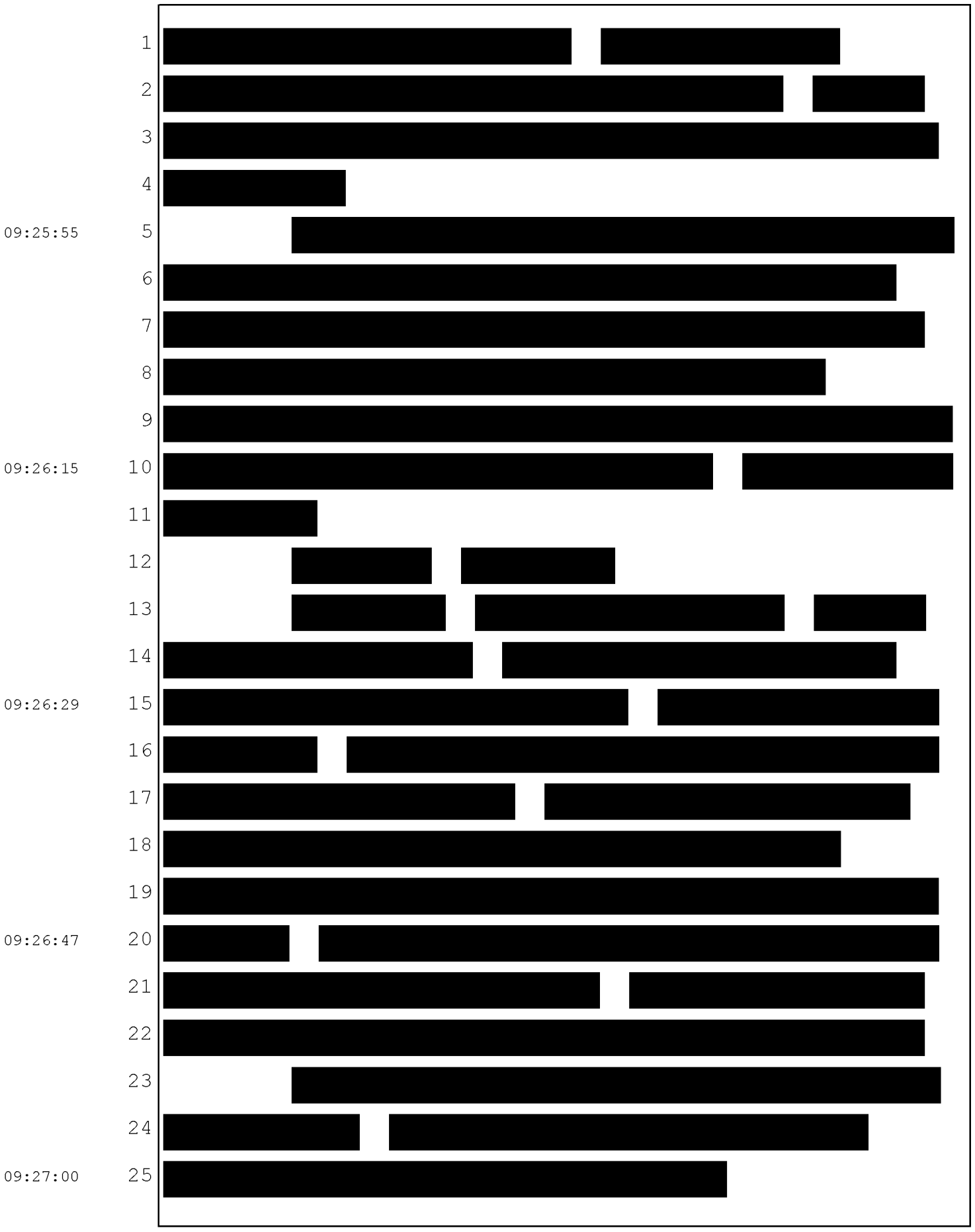
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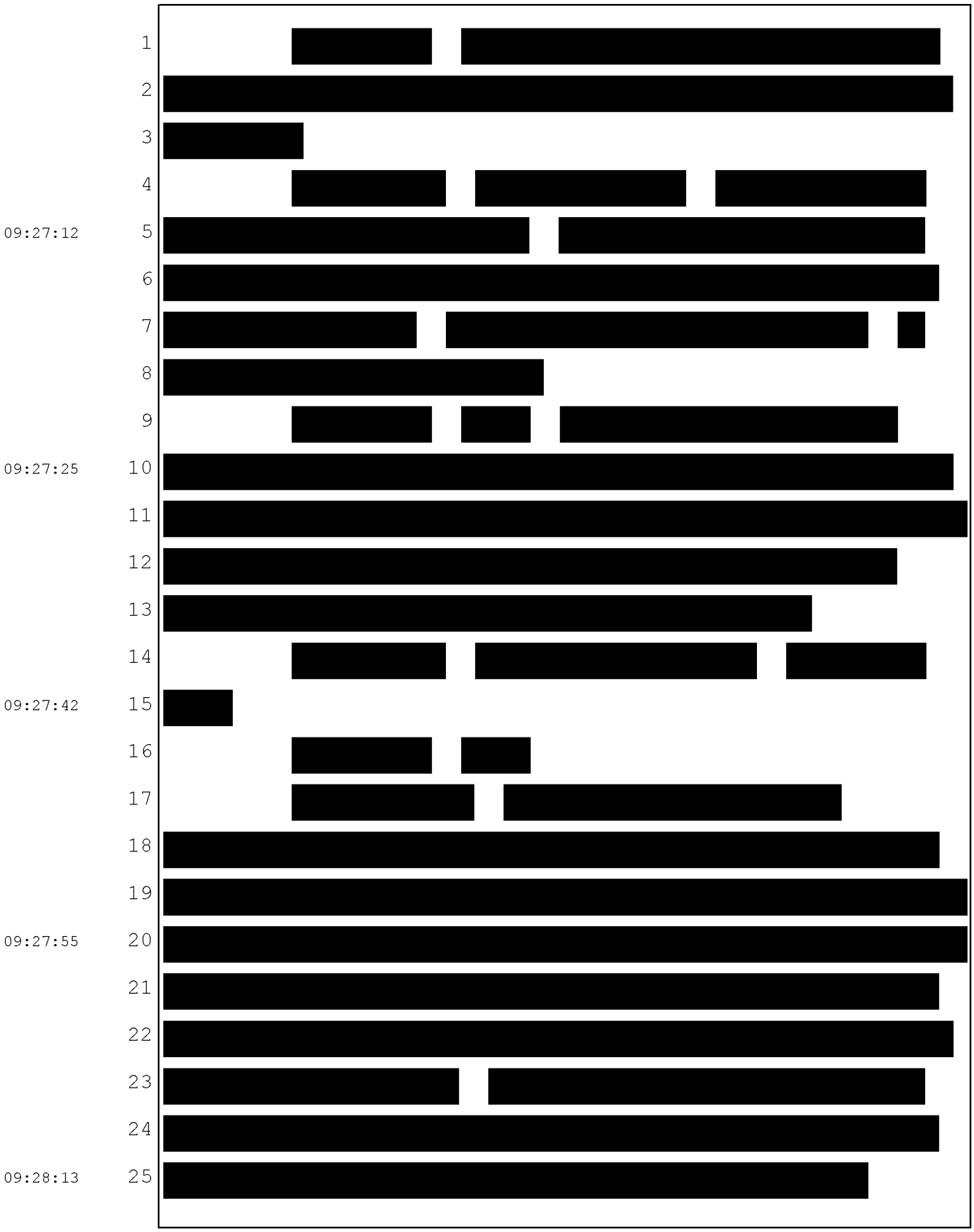
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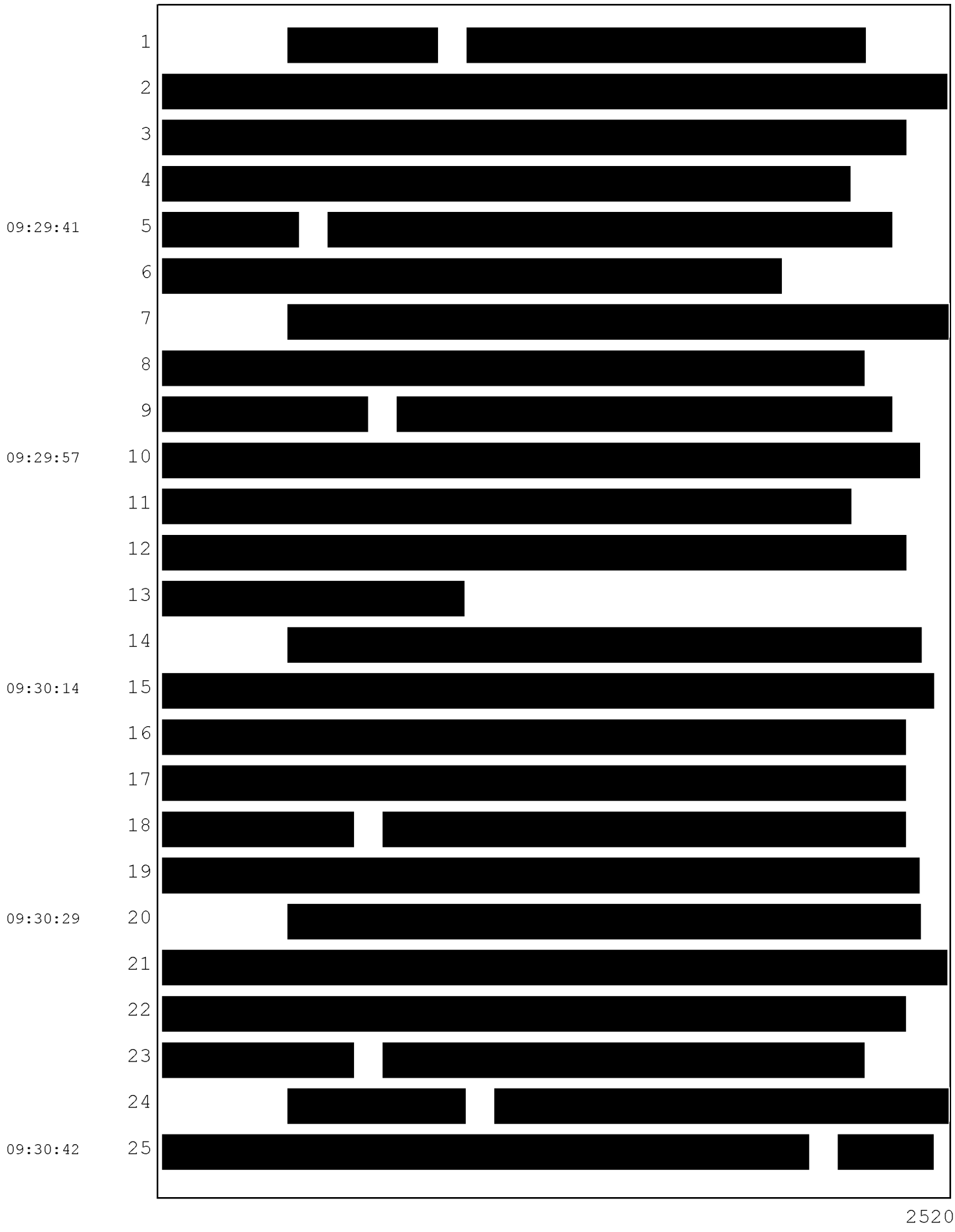
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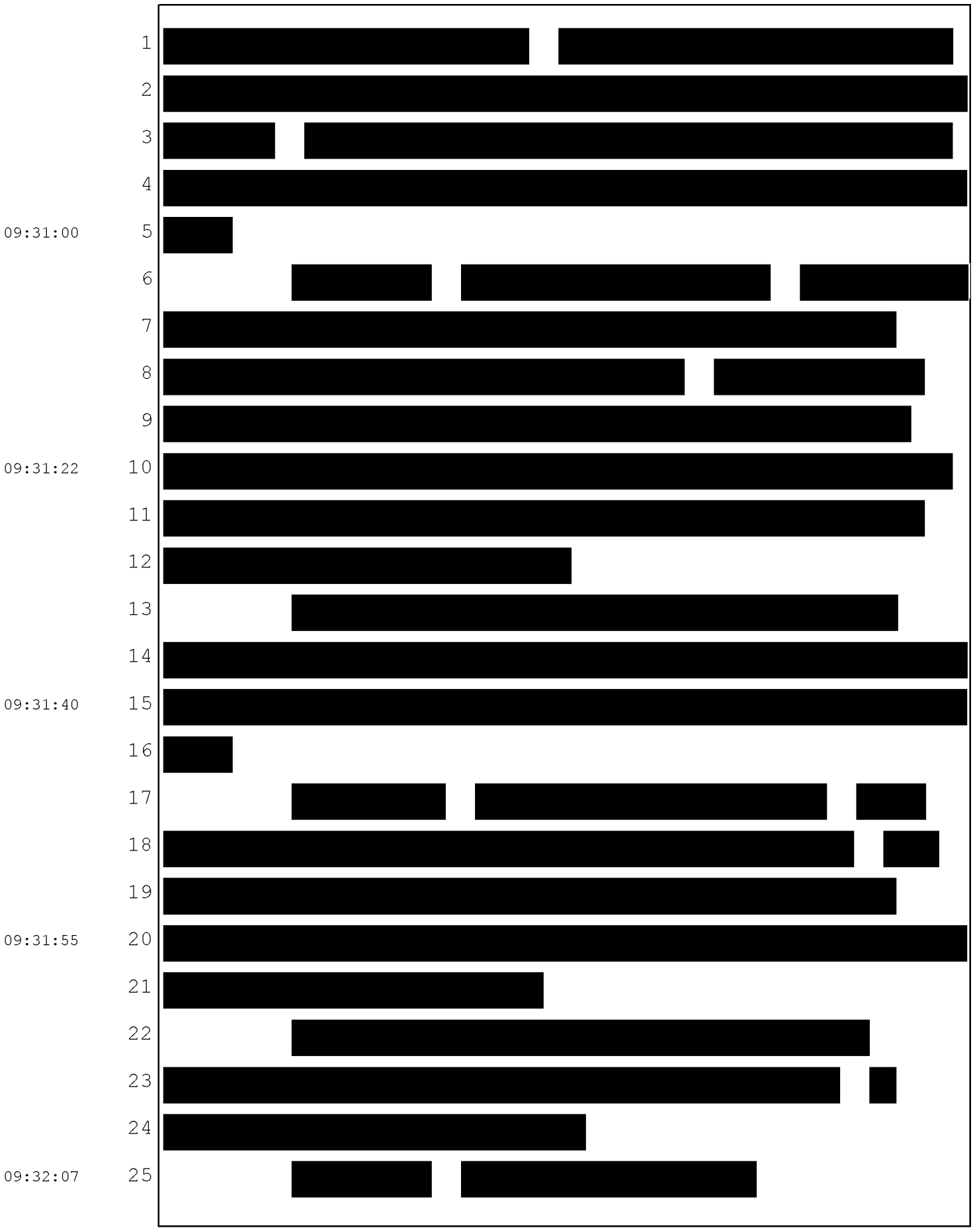
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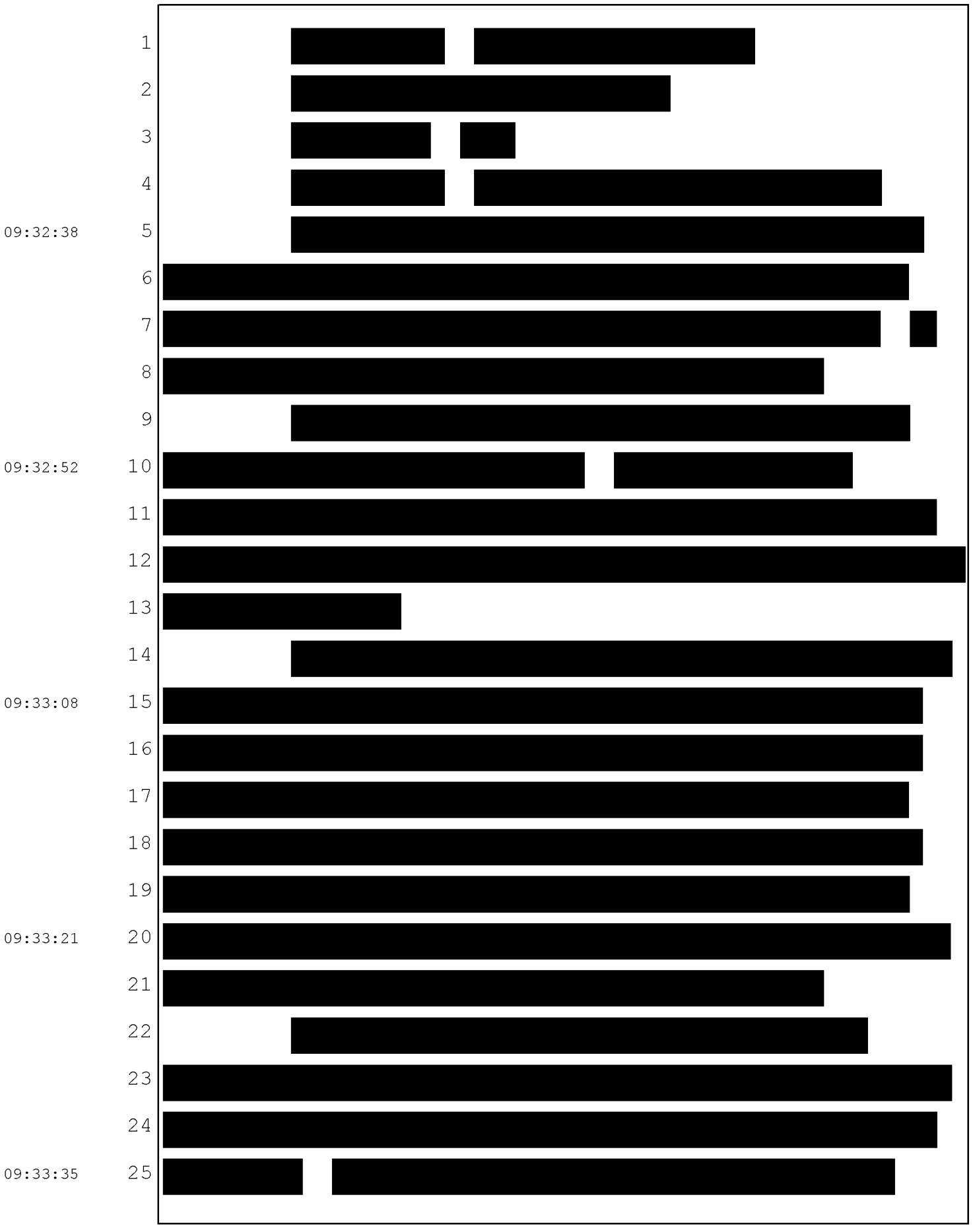
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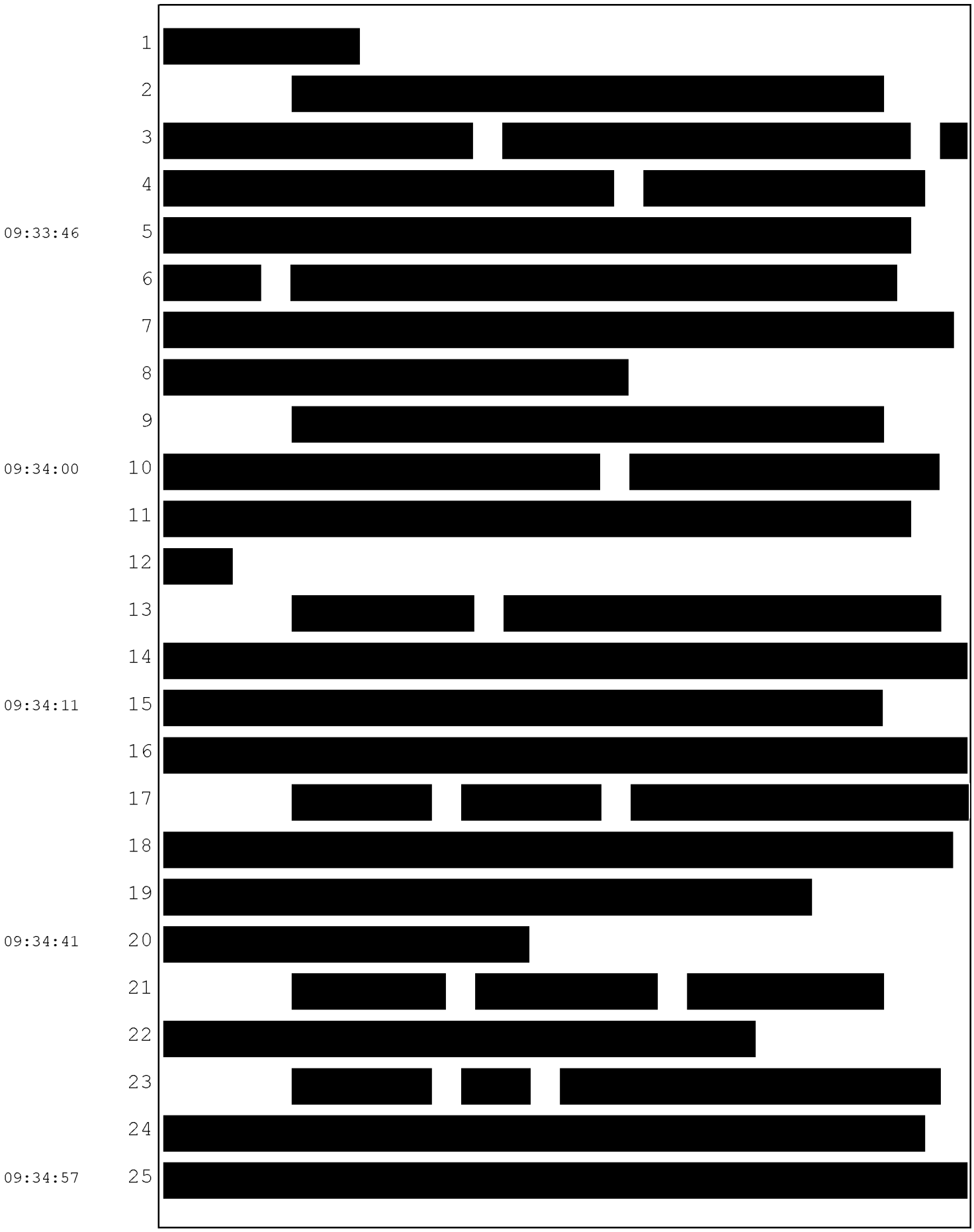
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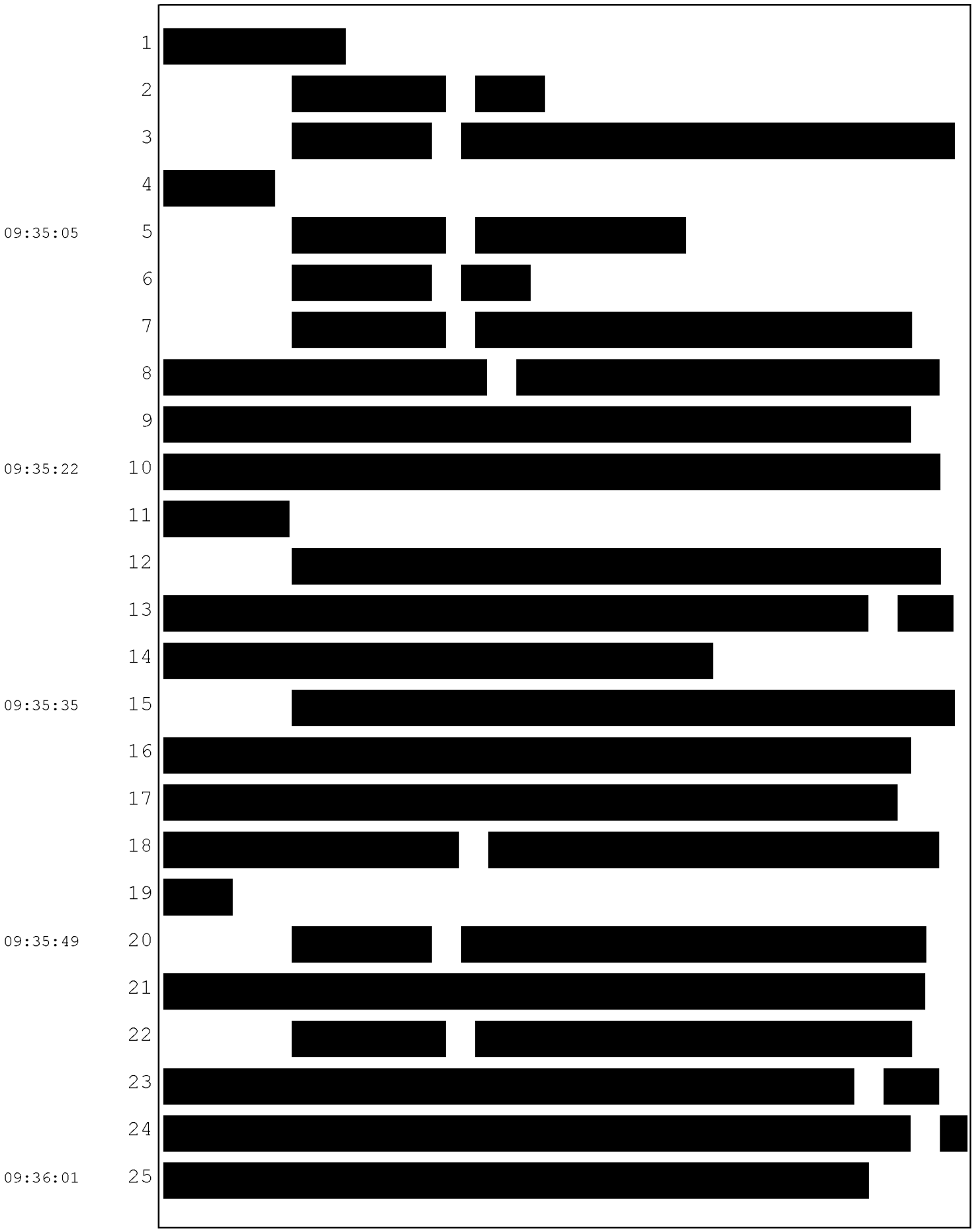
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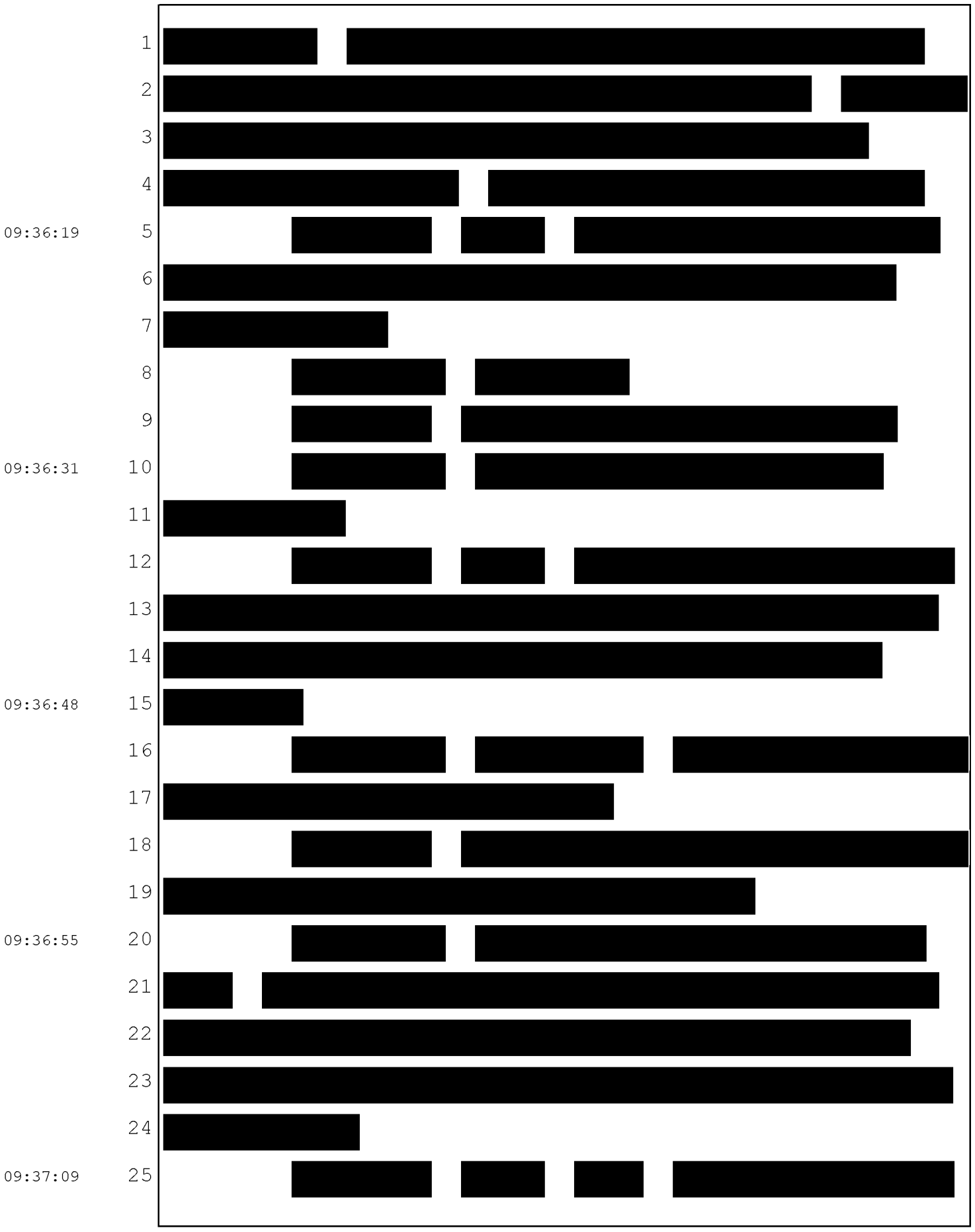
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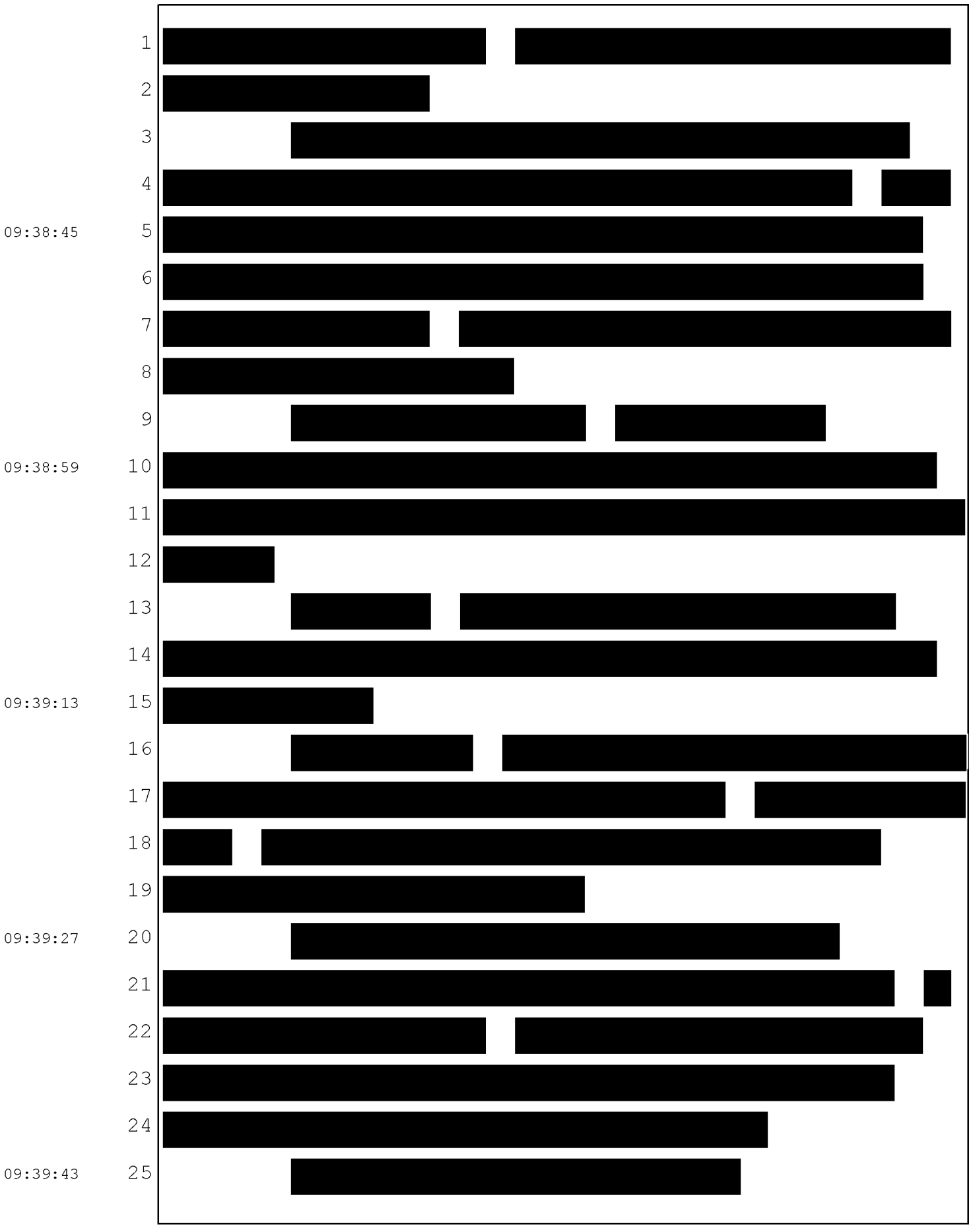
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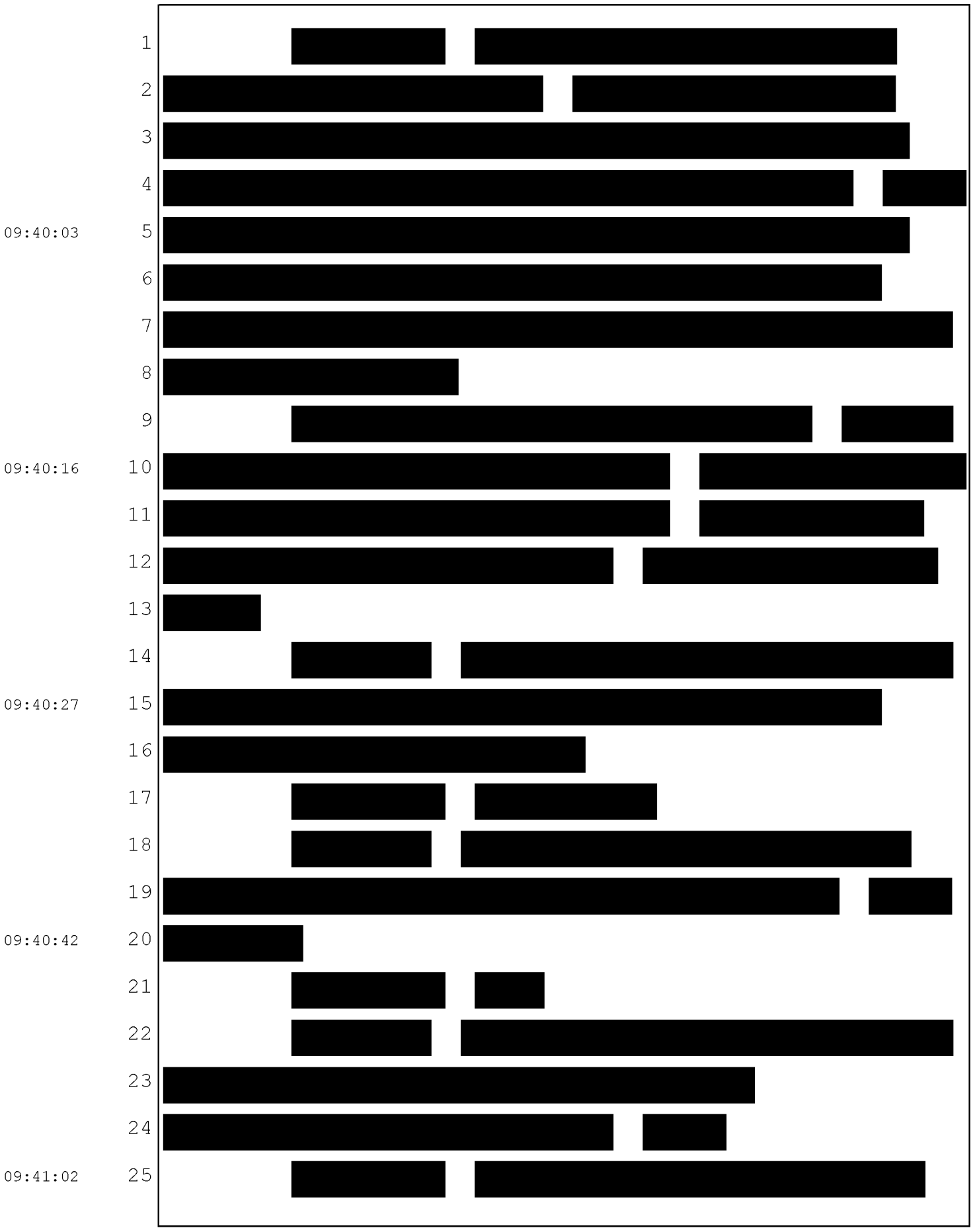
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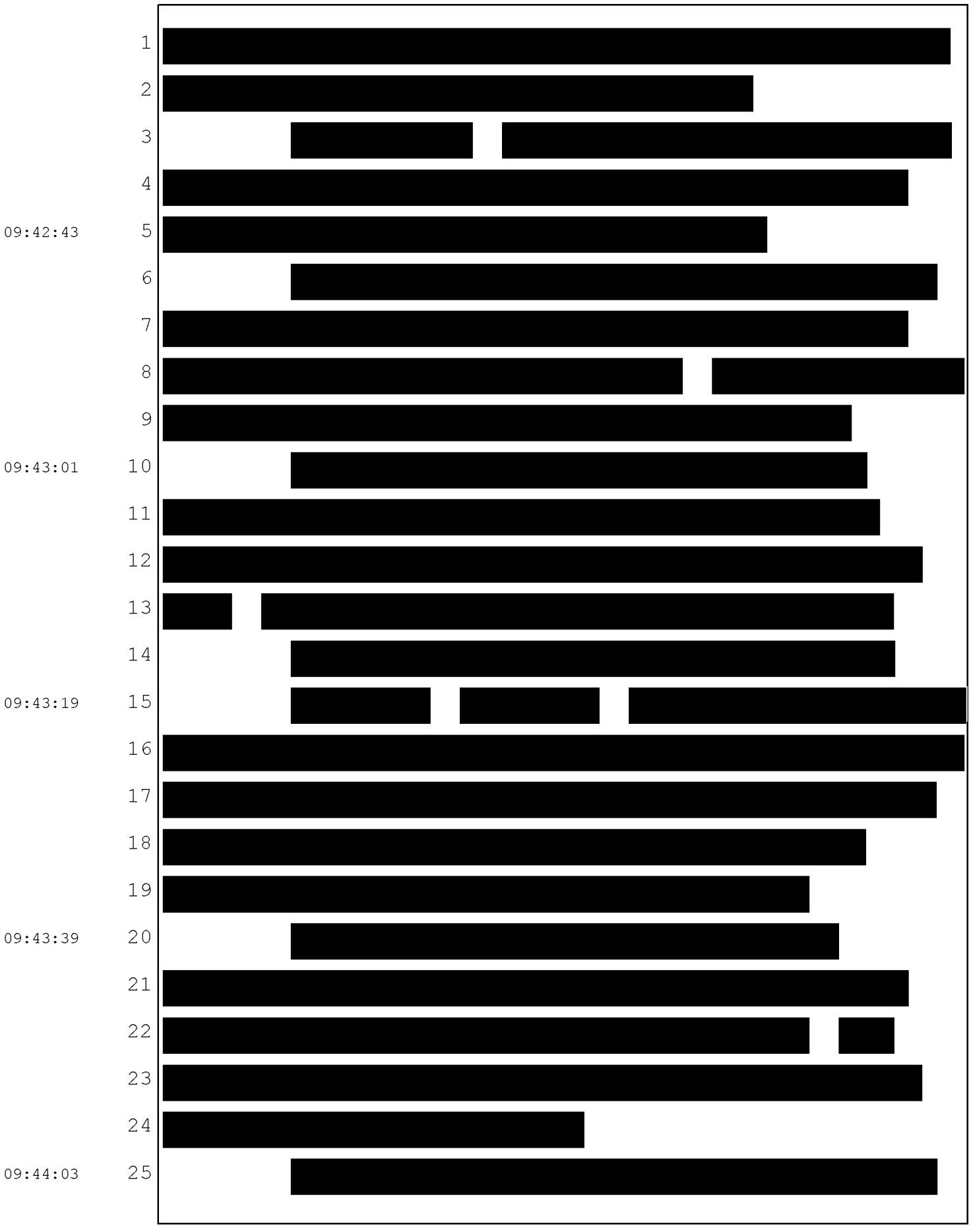
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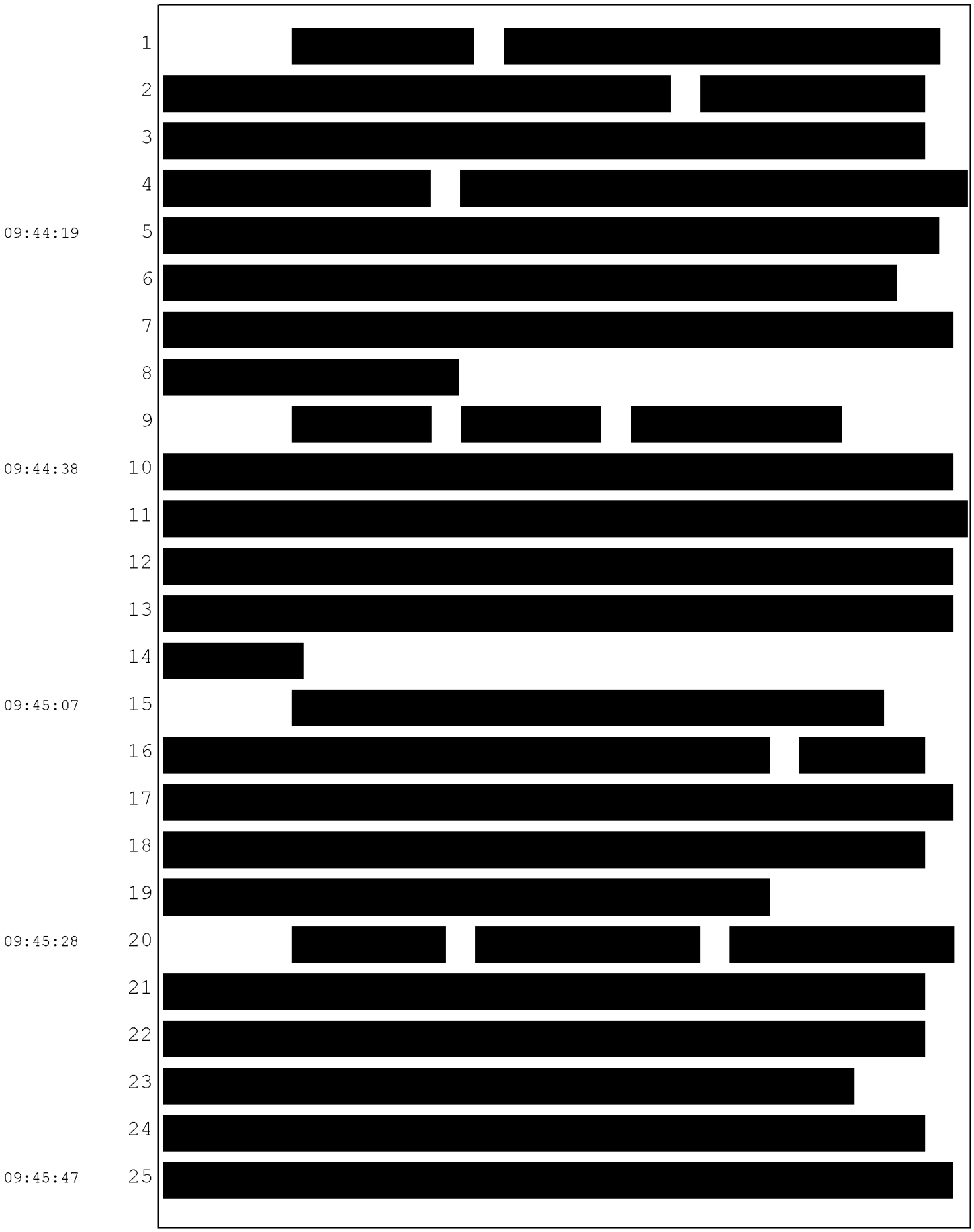
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(Jury enters courtroom.)

THE COURT: Good morning, Ladies and Gentlemen. Welcome back. We are now going to resume with the plaintiff's case.

Mr. Wisner, you may call your next witness.

MR. WISNER: Thank you, your Honor. At this time we call Dr. Alfred Neugut to the stand.

THE COURT: Good morning, Dr. Neugut. If you could please step up here to the witness stand and please remain standing while the clerk swears you in.

THE WITNESS: Thank you.

ALFRED I. NEUGUT,

having been first duly sworn, was examined and testified as follows:

THE CLERK: Would you please state and spell your name for the record.

THE WITNESS: Alfred I. Neugut, N-E-U-G-U-T.

THE COURT: Thank you.

You may proceed, Mr. Wisner.

DIRECT EXAMINATION

BY MR. WISNER:

Q. Good morning.

A. Good morning.

Q. How are you doing?

A. Good.

09:48:49 Q. Could you please introduce yourself to the jury.

A. Hi. My name's Alfred Neugut, N-E-U-G-U-T. I'm a medical oncologist and a cancer epidemiologist. I work at -- or I'm a professor at Columbia University in New York, and I've been on the faculty there since 1983.

09:49:11 Q. Now, Doctor, let's break that down a little bit. You said you're a medical oncologist. What does that mean?

A. Medical oncology is one of the specialties in medicine that takes care of cancer patients. It's the 09:49:27 one that's primarily involved with giving chemotherapy.

I've been treating cancer patients, well, actually going back to 1980, but as a specialty, and I've been Board-certified. I trained in it and I still see patients, which takes up now about I would say 25 to 09:49:50 30 percent of my time, seeing patients.

I specialize primarily at a center like my at Columbia, which is a major -- one of the major cancer centers in the United States, we all tend to subspecialize into specific cancers. So my specific 09:50:08 cancer from a treatment point of view is colorectal

cancer. I'm the main colon cancer person at Columbia, and I see about 35 to 40 cancer patients, mainly colon cancer, a week.

Q. When is the last time you saw a patient, Doctor?

09:50:25 A. Yesterday. I saw about 40 patients yesterday. They were are -- half of them are -- have potentially fatal disease and are -- have advanced colon cancer and are receiving chemotherapy or otherwise being treated for that.

09:50:40 The other half are patients who have had surgery for locally -- a localized cancer and are receiving or have received chemotherapy or otherwise being followed for the disease. And we follow them hoping that it won't recur, but if it reoccurs to be ready to treat them

09:51:01 further and to make sure that they do -- that they do okay.

Q. Where did you get your medical degree?

A. I was -- I went to medical school at Columbia. I've actually been at Columbia since 1968. I was an

09:51:17 undergraduate at Columbia. I went to Columbia College. I would say when I got into Columbia, our next-door neighborhood told my mother, "I'm sorry he couldn't get into an American school."

But subsequently I went to Columbia College, and

09:51:37 from there I went to Columbia Medical School. I was in

an M.D. Ph.D. program, which at the time actually -- I go back so long -- M.D. Ph.D. programs now are fairly widespread, but back then it was a fairly new thing. Because I was interested in research, you did research
09:51:54 and clinical training simultaneously.

So I did -- I spent two years in a laboratory doing molecular genetics and chemical carcinogenesis, studying the chemical carcinogens while I was in medical school. This again, we're now talking back in the
09:52:14 mid-'70s. This is between 1972 and 1977, when I did my training for medicine and my Ph.D.

I graduated in 1977 with a Ph.D. and an M.D. The Ph.D. was focused on the growth rates of cancer cells and also on looking at tumor initiators and promoters and
09:52:47 all of that.

From my perspective, the main thing I learned was that I was a klutz in the lab. Everything I touched in cell cultures got contaminated, and I learned that I was not cut out for the research laboratory. That was
09:53:02 not for me.

And -- but I did enjoy research, and I thought I was good at least in having ideas and thinking about research.

So when I graduated medical school, I went on to
09:53:15 do my clinical training. I went on to do a residency in

internal medicine from 1977 to 1980. I did that at the Albert Einstein College of Medicine in the Bronx in New York and was Board-certified in internal medicine.

At the same time, I -- I had gotten, because of my laboratory training and my other interests, I was fascinated by cancer and wanted to do further training both clinically and otherwise in cancer. So I decided I would train in -- and do clinical work in oncology.

So when it came time to go on from internal medicine, I decided to specialize in cancer and medical oncology.

By the way, can everyone hear me? I don't know.

So I ended up going for clinical training to Memorial Sloan-Kettering Cancer Center in New York, which is a cancer center. I trained there in clinical oncology, learned how to do chemotherapy and learned about cancer from a clinical perspective.

But at the time, from a research point of view, again, I didn't want to go -- everyone was pushing to go into a lab, but as I say, that wasn't to me. And at the time there was a relatively new area in what's called epidemiology, which I guess we'll talk more about shortly, but which didn't force me to go into the lab, where I could do mostly thinking and desk work. So I wanted to pursue epidemiology and Public Health, which --

which is what I pursued.

So I left Sloan-Kettering to go -- they did not have a strong program in that so I went back to Columbia -- actually, they recruited me for a new program
09:55:08 they had to train clinicians in Public Health and epidemiology.

So I spent two years at Columbia. I went back to Columbia, where I did a Master's degree in epidemiology, while at the same time finishing my
09:55:22 clinical training in oncology. Oncology, to do a sub-specialty in clinical, was three years.

So I finished my clinical training in medical oncology, was Board-certified in medical oncology, while at the same time doing a Master's degree in epidemiology
09:55:38 learning how you do epidemiology statistics and study design and things of that sort and learning how to do it.

At the time at Columbia -- again, now we're in the early '80s, '81 to '83 -- there was no person doing cancer epidemiology. I was basically the only one doing
09:55:57 cancer. Everyone was studying other things from a research point of view. But I did learn the methods, which is mainly what you have to learn in epidemiology, is epidemiology.

And so in essence, I became the cancer
09:56:13 epidemiologist at Columbia almost as the only one doing

it. And actually, in my second year as a student, there was a course in cancer epidemiology which I wanted to take. There was no one to teach it so I taught it. I enrolled in it, gave myself an A. And I got three
09:56:35 credits for it. And I've actually taught it ever since. So since 1982, I've taught the course in cancer epidemiology at Columbia.

And --

Q. Doctor, let me throw in some questions before I
09:56:49 get an objection.

A. Oh, I'm sorry. I was on a roll.

Q. Well, let me throw in a question. Okay. So I just want to backtrack. We've got a medical degree. We have a Ph.D. Is that pathology?

09:57:01 A. They call it pathobiology -- so because I don't actually do pathology in the sense of looking at slides and, you know, deciding what kind of tumor or something like that, but it's the study of abnormal human biology.

Q. And then you also have a Master's in
09:57:19 epidemiology?

A. Correct.

Q. All that is from Columbia; is that right?

A. Everything's from Columbia.

Q. So you've basically been a professor at Columbia
09:57:27 since?

A. So I joined -- so when I finished my Master's degree in Public Health, which was in 1983, and my clinical training, which was also in 1983, I finished my fellowship in medical oncology and was Board-certified, I got a joint appointment at Columbia in medical oncology and in epidemiology in the School of Public Health. And so I've had a joint appointment ever since I started as an assistant professor in medical oncology and in epidemiology. And then have gradually over the years risen through the ranks: Assistant professor, associate professor, full professor.

And I actually had a chair, you know, an endowed chair, in cancer research, which I've had since -- I don't actually know. I don't remember what year, but since about 2005, which is a special honor that's given for achievements in research and whatever.

So -- so I'm basically now my rank is -- or my title is Myron M. Studner, Professor of Cancer Research and Professor of Medicine in Epidemiology at Columbia. And then I have various other titles, but those are the main ones that I talk about.

Q. So Doctor, you kind of -- you said I have research and whatever. Let's talk a little bit about that.

How many peer-reviewed journal articles have you

published in the area of cancer and the causes of it?

A. Well, if you say "the causes of it," I'm not exactly sure how many papers. I've published over 600 papers. I would say of those papers, if we took just about causation, probably 3- or 400 have some tie to causation.

09:59:10

But I have other interests in terms of, for example, my research more recently, say since about 2000 or 2003, has been on quality of care for cancer patients. So I do a lot of research using epidemiologic methodology to study what's called health outcomes research, which is the quality of care patients get, who, if you're supposed to get chemo for certain type of -- in certain contexts, does everyone get it. If they don't get it, why don't they get it? Are they too poor? Are there racial disparities? Are there gender differences and things like that.

09:59:31

09:59:51

And again, this uses the same types of epidemiologic methodology as is used in etiologic research. So it's really an epidemiologic type of research. That's now one major area of my research efforts.

10:00:11

The other major area of my research -- my current research efforts is since about 2007 or 2008, for -- someone -- someone gave -- some agency gave money

10:00:32

to Columbia to study HIV and cancer -- or to study HIV and cancer in Africa, where the -- there's high rates of -- obviously of high prevalence of HIV in sub Saharan Africa. And no one else knew anything about it so they
10:00:54 asked me to do it.

And I didn't have any special interest earlier in that, but I started looking into it. So since then I've been actually studying HIV and the effect of HIV preval- -- the prevalence of HIV in South Africa is about
10:01:17 20 percent.

So how a person having HIV affects their cancer natural history, how it affects their treatment, how it affects their outcomes.

Cancer rates are going up in sub Saharan Africa
10:01:33 because we're now giving out antiretroviral therapy to people with HIV. Before, the life expectancy in South Africa or in other countries in sub Saharan Africa was very low because of HIV. So people weren't living past 50 or 55, and so they weren't getting the cancers that we
10:01:52 normally associate with in the West -- breast cancer, colon, prostate. They didn't live long, and those are diseases of middle and older ages, but now they're getting them more so it's becoming a major problem in sub Saharan Africa. And so we're studying how that interacts
10:02:07 with HIV.

I have now about several million dollars in research money to study breast cancer, prostate cancer, and lung cancer in sub Saharan Africa.

Q. All right, Doctor. You know, we could go through your CV for literally two hours, but we have a two-hour clock here. So I'm just going to put it together.

MR. WISNER: Permission to publish Dr. Neugut's CV to the jury.

10:02:30 THE COURT: Any objection?

MR. LOMBARDI: No objection.

THE COURT: Very well.

Q. BY MR. WISNER: All right, Doctor, we're looking here on the screen. This is a copy of your CV. We can 10:02:36 zoom in here. This is actually a bit old. It's as of April 1st, 2017.

Do you see that, Doctor?

A. Yes.

Q. It goes through all your -- just to give you 10:02:44 some context, Doctor, my résumé is one page. So let's go through this quickly.

So we have your licenses. We have hospital appointments. And this reflects the various places you're allowed to practice medicine; is that right?

10:03:00 A. Yes.

Q. Okay. Honors and awards, these are various things that you've received through your professional career; is that right?

A. Yes.

10:03:06 Q. And I know this stops at 2016. I understand you recently achieved -- received a lifetime achievement award; is that right?

A. Yes, uh-huh.

Q. And what was that award and who was it from?

10:03:18 A. So the American Society of Preventive Oncology is the leading cancer epidemiology organization in the United States. Actually, I was president of it at one point years ago.

So two years ago they gave me their 10:03:33 distinguished achievement award, which is given out every year to I guess someone who has had distinguished achievement.

So it's like their singular honor every year. You give a talk. No money, but you get a little plaque. 10:03:48 Somewhere it's hanging in my office, but whatever.

Q. And we have grants here. And this portion of your CV goes on and on and on. Doctor, can you give me a ballpark of the amount of grant money that you've received for research cancer?

10:04:08 A. I would ballpark it at around somewhere in the

45 to $50 million range.

Q. And these are grants from various governmental and private institutions to have you research cancer and the causes of it; is that right?

10:04:20 A. Yes, all related to cancer.

Q. And then your sort of administrative responsibilities. This reflects like, what, your supervisory responsibilities; is that right?

A. Yes.

10:04:30 Q. And then we have your teaching experiences, and as you can see right here, you discuss the cancer -- cancer epidemiology that you've been teaching --

A. Uh-huh.

Q. -- since I guess it was created?

10:04:43 A. Uh-huh.

Q. All right. And then we keep going down here, and you have graduate student supervision.

Do you see that?

A. Yes.

10:04:50 Q. And so these are people you've supervised for their doctoral as well as Master's theses?

A. Yes.

Q. And then you have a bunch of post-doctoral training that starts here at the bottom of page 10.

10:05:02 Do you see that, Doctor?

A. Yes.

Q. It goes on for a few pages. And what is a post-doctoral training? What are you doing in that context?

10:05:08 A. Post-doctoral training is someone who has gotten a Ph.D. and In the past -- go back, I don't know, 10 or 15 years, maybe after a Ph.D. you could actually get a job. Nowadays that's becoming more difficult.

So people go on and do two or three years of 10:05:30 what's called post-doctoral training to get more -- more experience and to get some papers under their belt and to get an opportunity to get a faculty position or to do something else, but usually they'll get a faculty position.

10:05:45 So they do a post-doc, working with some senior or someone at some academic institution to do more research or maybe something different so they get a little more experience and then go on to do that.

And so I've been lucky enough to have a lot of 10:06:03 post-docs train with me, and they've all -- many of them have gone on to have their own academic -- successful academic careers.

Q. All right. I'm going to sweep through this. You have you've worked on an editorial board. So I take 10:06:21 it you've reviewed other people's published literature to

see if it was appropriate?

A. We see manuscript reviewers. So, you know, we do peer review. That's if you publish a lot of papers, they're also going to ask you to do peer review for other people's papers. So I end up doing a lot of peer review for various journals.

Q. I'm sorry, Doctor, are each one of these journals one that you've been a peer reviewer on?

A. Yes.

Q. Oh, look at that.

And then there's different committees you've served on. For example, I see you served on a Federal National Cancer Institute Committee.

Do you see that?

A. Yes.

Q. And these are various things that you've done as part of your academic and research career; is that right?

A. Yes.

Q. You have state and local. We have study sections. We have private foundations, international, other, and then we start the publications.

Do you see that's on page 20?

A. Yes.

Q. Okay. And then this goes on, I mean, for hundreds. So let's go through this quickly. All right.

So that gets us through publications. We're on page 61 now, invited reviews. What is an invited review?

A. Sometimes a journal will ask you to -- actually will invite you to write a chapter or a review article on some subjects. So opposed to you having to write it up yourself and then submit it and it will be accepted or whatever.

10:07:38

Q. Okay. We have a few of those, and we have book chapters, quite a few of those. It goes on to page 65 with editorials. It goes on for a few pages. We have books and letters.

10:07:55

Let's take a second and just talk about these books. One of these stands out in particular. It refers to the health effects of herbicides in Vietnam.

Doctor, have you looked at herbicides and its effects on cancer?

10:08:09

A. I'm sorry.

Q. Have you looked at herbicides and its effect on cancer in your work and research?

A. So I don't myself study it as a researcher, but this book reflects a committee that was established in the early '90s by the Institute of Medicine. It was asked by the Veterans Administration at the time to review for the purpose of deciding whether a Vietnamese veteran -- not Vietnamese, but --

10:08:17

10:08:46

Q. Vietnam.

A. Yes. That veterans of the Vietnam War, who were exposed to Agent Orange, there was literature that suggested that because of exposure to Agent Orange, they were at an increased risk of cancers and other diseases, other problems.

10:09:01

So the VA was interested or wanted to know whether they were obligated to compensate them for these injuries stemming from Agent Orange exposure from the herbicide spraying in -- that took place in Vietnam. So they established this committee of experts, not dissimilar to the -- it was actually very similar to the way the IARC committees are established.

10:09:16

So I was on that committee. I was the chair of the cancer subcommittee within this -- within this committee.

10:09:35

So the subsequent report was actually published as this book in 1994 and found that there were certain cancers that stemmed from being exposed to Agent Orange and that -- and I guess the recommendation was that the VA should compensate Vietnam veterans who were exposed to Agent Orange and developed these specific cancers subsequently.

10:09:55

Q. All right. Then we have letters, invited presentations. That goes on for a few pages. And that's

10:10:10

the end, page 75 of your CV?

A. Uh-huh, yes.

MR. WISNER: At this time, your Honor, I'd like to have Dr. Neugut recognized as an expert in the area of medical oncology and cancer epidemiology.

10:10:24

MR. LOMBARDI: No objection.

THE COURT: Any *voir dire*?

MR. LOMBARDI: No objection, your Honor.

THE COURT: Very well. Then I will accept Dr. Neugut as an expert in the areas of medical oncology. And Counsel, did you ask for cancer epidemiology?

10:10:32

MR. WISNER: Yes, your Honor.

THE COURT: And cancer epidemiology. All right. Thank you. You may proceed.

Q. BY MR. WISNER: All right, Doctor. What were you asked to do in this case?

10:10:50

A. I'm sorry.

Q. What were you asked to do in this case?

A. I was asked to review the literature and to opine on the association between Roundup or glyphosate and its association with non-Hodgkin's lymphoma.

10:11:03

Q. And when you were asked to do this, what's the first place you looked at to see if there was an association?

A. So when I was -- so whenever I'm asked to be an

10:11:21

expert or to comment on a case, I do my own literature review initially before I will accept a case, because obviously I don't want to participate in a situation where I'm not comfortable or where I don't know that it's
10:11:49 true or whatever. So I usually do sort of my own literature review off the record sort of uncompensated.

So I usually do a literature review by using published papers, and if there is an IARC publication on it, I usually start with IARC, or at least the IARC
10:12:16 publication is a major part of my initial assessment.

Q. Why is that? Why do you go to IARC, Doctor?

A. I would say that within the scientific and academic cancer community, IARC is recognized as the main arbiter of -- the prime arbiter of what constitutes a
10:12:40 carcinogen or a cancer-causing agent. I would have trouble even naming a second -- I would have trouble naming a second choice.

Now, you do have sometimes the problem that IARC hasn't reviewed -- you often have the problem that IARC
10:12:57 hasn't reviewed a particular agent, and then obviously in that context, you don't have an IARC Monograph or an IARC publication to use as a -- to help you.

But when there's an IARC Monograph, then I would say almost uniformly that's what everyone -- and I can't
10:13:22 speak for everyone; I can speak for myself and I can

speak for my colleagues who I know or for most of the academic community -- IARC is usually the main arbiter of what a cancer-causing agent is.

Q. You don't go to the EPA?

10:13:36 A. No.

Q. Why not?

A. It never crossed my mind.

Q. Okay. Now I want to talk to you briefly about what epidemiology is. And I understand you actually have

10:13:48 prepared a demonstrative to help the jury sort of understand conceptually what it is; is that right?

A. I don't know if it's to tell them what epidemiology is, but I can tell them what epidemiology is if you like.

10:14:01 Q. Okay. Well, hold on a second.

MR. WISNER: Permission to publish exhibit -- I don't believe it's stamped, but this demonstrative.

MR. LOMBARDI: No objection, your Honor.

THE COURT: Very well. You may proceed.

10:14:13 Q. BY MR. WISNER: The reason why I have not described this demonstrative correctly is because I actually don't understand it.

A. You're going to have to go sit with the jury.

Q. And walk us through what this document is and

10:14:25 how -- come down here. And walk us through exactly what

this is and how to understand it and actually see if I can turn it so everyone can see it, your Honor.

A. Can you?

Q. Here's a marker if you need it.

10:14:40 A. Thank you. So before I even address this, let me tell you what epidemiology is more broadly before I address the -- because this is really more intended to tell you how you do epidemiology than what epidemiology is.

10:14:57 So epidemiology comes from the word "epidemic," and epidemic, as you probably know, is an excess of a disease in the population and -- over a time frame, when you have too many cases of flu or whatever in a given population over a given time frame. And that's where the 10:15:20 word "epidemiology" came from.

When I started out everyone thought -- epidemiology wasn't as well known. Everyone thought it came from the word epidermis, and they thought I was studying skin. But in truth, epidemiology is what I just 10:15:33 said.

And in epidemiology, what we study is the distribution of diseases, its incidence, how common it is, and things like that, but its incidence over time and things like that.

10:15:47 But the purpose of epidemiology is for Public

Health purposes. It's not so much intended for medicine for treatment, but for Public Health in order to reduce its incidence in the population, in order to prevent the disease from occurring. If we figure out why you get the disease, then we can take steps to prevent the disease.

So the purpose of epidemiology, the underlying purpose, is to figure why people get heart disease, colon cancer, whatever disease, HIV, et cetera, to figure out why they get it. And to do that is not -- often not very easy to achieve and through various methodologies.

So going to this board that sets us up for this, so in most of science, in fact -- this is a pretty good layout for science -- we're mostly studying in most of science is the association between an exposure and an outcome. Does tobacco smoking cause lung cancer? Does taking Lipitor reduce your incidence of heart disease? Does putting salt in a solution increase the release of heat from a solution?

This is the underlying -- you could say this is the underlying phenomenon of science. This is for all of science. But in the context of epidemiology, we're -- what we're talking about today, the exposure and the outcome is usually some exposure, whatever it might be -- obviously, in our case we're talking about Roundup or glyphosate -- and an outcome, which in our case today of

course is non-Hodgkin's lymphoma. So, but again, you could put in any two things.

And as in all of science -- as in all of science -- and epidemiology is no different than all of
10:17:53 science -- we start off with what's called the null hypothesis. And to put it in CSI terminology, or Law and Order, which is my favorite show, we start off innocent until proven guilty. We assume that they're random. In other words, the underlying assumption is that the
10:18:14 exposure and the outcome have nothing to do with each other.

And it's our job, our duty or whatever you want to call it, our underlying goal is to assess, to do studies to determine whether the exposure and the outcome
10:18:29 have something to do with each other, they're nonrandom document. But the underlining is we start off everybody's innocent until proven guilty.

And then it's our job to find evidence to assert that they're nonrandom.

10:18:43 So the studies that we do in epidemiology are to find nonrandomness or to see if there is nonrandomness, if the two really are linked together.

A priori, we say tobacco and lung cancer have nothing to do with each other, but when we do studies and
10:19:01 we find that they are linked to each other, then we say

ah-ha, there's something going on. That's not necessarily causal. First we want to go see that they're linked to each other, that they're not random with each other.

10:19:09 So what an epidemiologic study does is, an epidemiologic study does not tell you that an exposure causes an outcome. An epidemiologic study tells you that the exposure and the outcome are statistically associated and nonrandom, that they occur together more commonly

10:19:26 than -- than would generally be the case in a general -- randomness would have asserted.

It doesn't mean they're causal. It just means that they're statistically associated more. They occur together more commonly than -- smokers and lung cancer

10:19:40 occur together more frequently than we would expect. It doesn't mean tobacco -- maybe having lung cancer makes you want to smoke because it makes your lungs feel better, for all you know. Could be. But just the fact is it's nonrandom.

10:19:54 And how do we do this in the epidemiologic study? Because only two types. Easy peasy. There's only two types of epidemiologic studies that exist. There's only two types. Easy. You either go this way or you go this way. No other study.

10:20:13 So if you start from the outcome, you say I'll

take people with -- I'll take smoking and tobacco because that's one we all know and can accept. I take a hundred people who smoke and a hundred people who don't smoke.

Q. 100 people with lung cancer.

10:20:34 A. I take a hundred people who have lung cancer, the outcome, I take a hundred people who don't have lung cancer, and they ask how many of you smoke, and if it turns out there's a large number in the lung cancer who smoked than in the control group, in the non lung cancer 10:20:51 group who smoked, and statistically that turns out to be different, then I assume or I see that lung cancer and tobacco are statistically associated.

So that's one type of epidemiologic study. That's called the case-control study. That started with 10:21:12 cases, lung cancer cases and control, people who don't have lung cancer. And that's called a case-control study. I started from the outcome, from the lung cancer, and I went back to the exposure. I asked how many smoke and how many don't smoke.

10:21:28 The equivalent in our study would be to start with people had have non-Hodgkin's lymphoma and people who don't have non-Hodgkin's lymphoma --

THE COURT: Excuse me, Doctor.

Mr. Wisner, can I please remind you to proceed 10:21:41 by way of question and answer.

MR. WISNER: Oh, sorry.

THE WITNESS: Sorry. That's his job.

THE COURT: And Doctor, if you're done using the demonstrative, perhaps you can return to your seat.

10:21:53 MR. WISNER: I think we have to do one more direction and he'll sit down.

THE COURT: Okay, great.

THE WITNESS: Yeah, one more minute and I'll be done.

10:21:58 Q. BY MR. WISNER: All right. So we'll talk about how this relates to NHL in one second.

A. Okay.

Q. But let's get onto the other type of study.

A. Right. So the other study is going forward, 10:22:07 which is to start from people who smoke and people who don't smoke and go forward to asking -- starting with people who smoke and people when don't smoke, and ask how many of each of them, if I take a large number of each, and ask how many of them get lung cancer in each group.

10:22:24 And then if the smokers get a higher rate of lung cancer than the nonsmokers, then we see that they're statistically associated because smoking and lung cancer is associated.

And again, I'll let the attorneys ask about 10:22:40 whether -- how that relates to our current case. And

that's called a cohort study.

So basically there's a cohort study or a case-control study, either going from the exposure to the outcome or from the outcome back to the exposure.

10:22:58 That's all of -- but again, these -- I'll sit down, your Honor.

Either one of these studies, the case control or the cohort study, on its own only tells us that there's a statistical association between the two; that it's not,

10:23:16 as I said, nonrandom between the two. It doesn't tell us if there is actual -- or that we might assume there is if it's very dramatic, someone might want to infer that. But on its own, it only tells us -- it only tells us there's a statistical association.

10:23:35 Q. Now, Doctor, the ability to conduct either a cohort or case-control study, is that in any way affected by the amount of exposure and/or the rarity of the disease outcome, here cancer?

A. So for that I have to riff a little bit on the

10:23:55 question of how you establish causation for a moment, which is -- it is harder -- the more rare the outcome is, the more rare the exposure is makes it more difficult to establish an association. And certainly if the two of them are uncommon, it becomes much more difficult to

10:24:24 establish these associations just statistically and

methodologically.

And if I can illustrate that, I'll say, for example -- well, let me back up and talk a little bit about causation because there you can see it a little better, if I may.

Q. Please.

A. So the whole question of how to establish causation is an issue. So how to establish causation goes back to Hippocrates or the Greeks, who talk about it, and all the medieval philosophers talk about how to establish causation. Establishing causation is a very difficult phenomenon over and above statistical association. And the question is how do we establish causation in general in our lives.

The answer is in a weird way. It's by -- generally speaking, it's by what I would call inductive reasoning. A child goes around, a toddler, and flips a light switch, let's say, in the kitchen, and the light goes on. The child doesn't necessarily put flipping the light switch with the light going on. Doesn't necessarily make the connection. But if he does it two, three, four, five times, after awhile, and every time the kid flips the light switch, the light goes on. After four, five, six times and the kid's start enough, if it's your kid, the kid will understand finally that flipping

the light switch makes the light go on, and he understands there's a causal connection between flipping the light switch and the light go on.

Now let's say we want to play with the kid's head, and we'll make it that randomly the light will only go on 50 percent of the time when the light switch goes on and it will be random.

So now the kid flips the switch. Sometimes the light goes on, sometimes the light doesn't go on. And the kid's going to be, like, uh-huh.

And so it's going to take now instead of maybe four or five or six times, maybe it will take eight or ten or twelve times, but after ten, twelve times, if it's only 50 percent, the kid's finally going to understand still that there's still a causal connection between flipping the light switch and the light going on, although there's something screwy about the light switch and the light going on.

Let's say, I made it one in ten times, one in ten times when you flip the light switch. We can do this, you know, and drive someone nuts.

But let's say you do it one in ten times, the light's randomly going to go on. It's a kid, it's a five-year old is going to figure out that the light switch is connected to the light going on. It would

actually be really difficult, it may take a really long time. Maybe the kid will figure out, maybe he won't.

So as you make it more and more unlikely of the probability of the light going on, more and more uncommon, it's going to be harder and harder for a child to establish a causal connection between the phenomenon and the outcome -- between the exposure, the flipping the switch, and the outcome.

10:27:18

Q. Now, Doctor, you're talking one out of ten. We're talking about cancer like non-Hodgkin's lymphoma --

10:27:33

A. Right.

Q. -- what numbers are we talking about here?

A. So most of cancer epidemiology, if you actually look at the literature and you see what I do every day or most of my colleagues do every day, is focused on the four most common cancers, which are breast, prostate, colon, and lung. They occur in an incidence rate of one in a thousand per year. And so most studies in colon cancer -- in cancer will focus on those four cancers, which are very common.

10:27:46

10:28:03

If we talk now about -- and even there, the studies are very conflicting and not that easy to do and to establish causal association.

If we talk about non-Hodgkin's lymphoma, the topic of the day, then we're talking about two in 10,000

10:28:16

per year, which is a very uncommon cancer, which makes it much more difficult to study.

And if I put on top of that that we're talking now about -- at least in the context of Roundup -- a relative risk of about 1.4 to 1.5, so we're talking about a 50-percent increase of the incidence of the disease. So we're talking about going from two in 10,000 to three in 10,000.

So that is a very difficult phenomenon to establish using epidemiologic methodology. And that is why -- that's in part why we're here today, but that's a very difficult thing to establish through epidemiologic studies, but not impossible. And it's difficult and makes it -- it means that any -- a small error, any small phenomena are going to have a profound effect on what you observe in the epidemiologic studies. Because we're talking about a very relevant natively small delta -- relatively small difference to be observed.

And to try to establish with evidence, as I said before, innocent -- the entire epidemiologic methodology is conservative. All of science is conservative. We don't want to find a positive finding when there isn't one. We don't want to implicate an innocent person, an innocent exposure, as being guilty when it's not.

So the entire statistical and epidemiologic

methodology is constructed in such a way as to bias the -- the outcomes to be no or negative unless there's really a true positive relationship or true association.

And so when you don't see a positive association, you cannot be certain if you don't see the association, either because it's truly innocent, truly negative or null, or because all the biases have made it null or biased the results in that direction.

When you see a positive finding, then you can probably have a lot more confidence because, again, we're more -- we've set the system up to make the positive finding the more robust phenomenon.

Q. All right. Doctor, I want to talk to you about those biases for a second.

MR. WISNER: Permission to approach the witness with the binder.

THE COURT: Yes.

MR. WISNER: Here's your binder. Don't worry, we're not going to use all the stuff in there.

THE WITNESS: Okay.

Q. BY MR. WISNER: But I do want to draw your attention to Exhibit 682, which should be in your binder under the tab 682.

A. Okay.

Q. And this is a journal article. First author is

Aaron Blair, and this is something you've reviewed; is that right?

A. Yes.

MR. WISNER: Your Honor, permission to publish.

10:31:51 THE COURT: Any objection?

MR. LOMBARDI: Just checking something, your Honor.

Your Honor, we do have an objection as to not having been identified on a reliance list.

10:32:10 THE COURT: Do you wish to approach, Counsel?

MR. WISNER: Here (indicating).

(Sidebar.)

[redacted]

10:33:02 [redacted]

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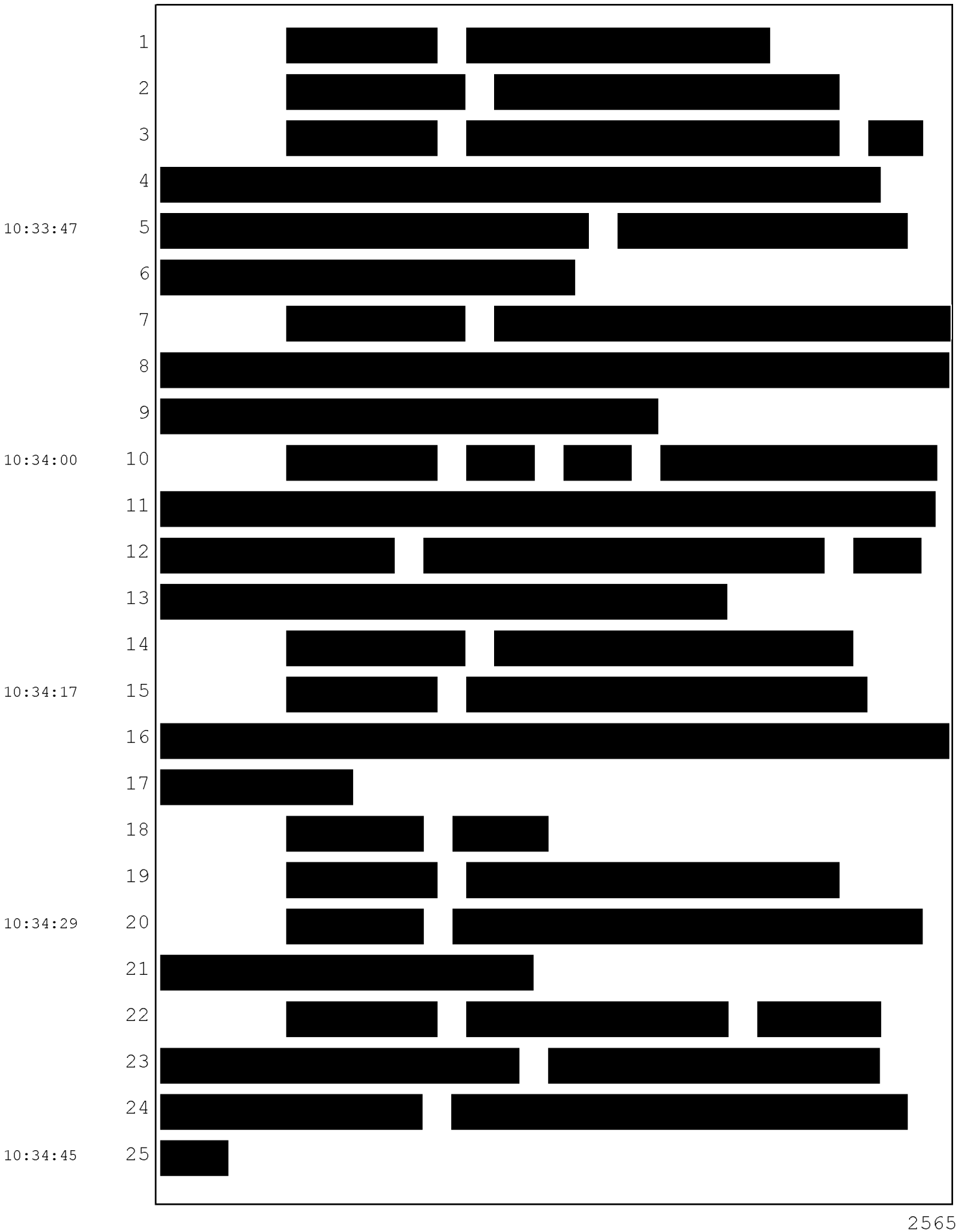
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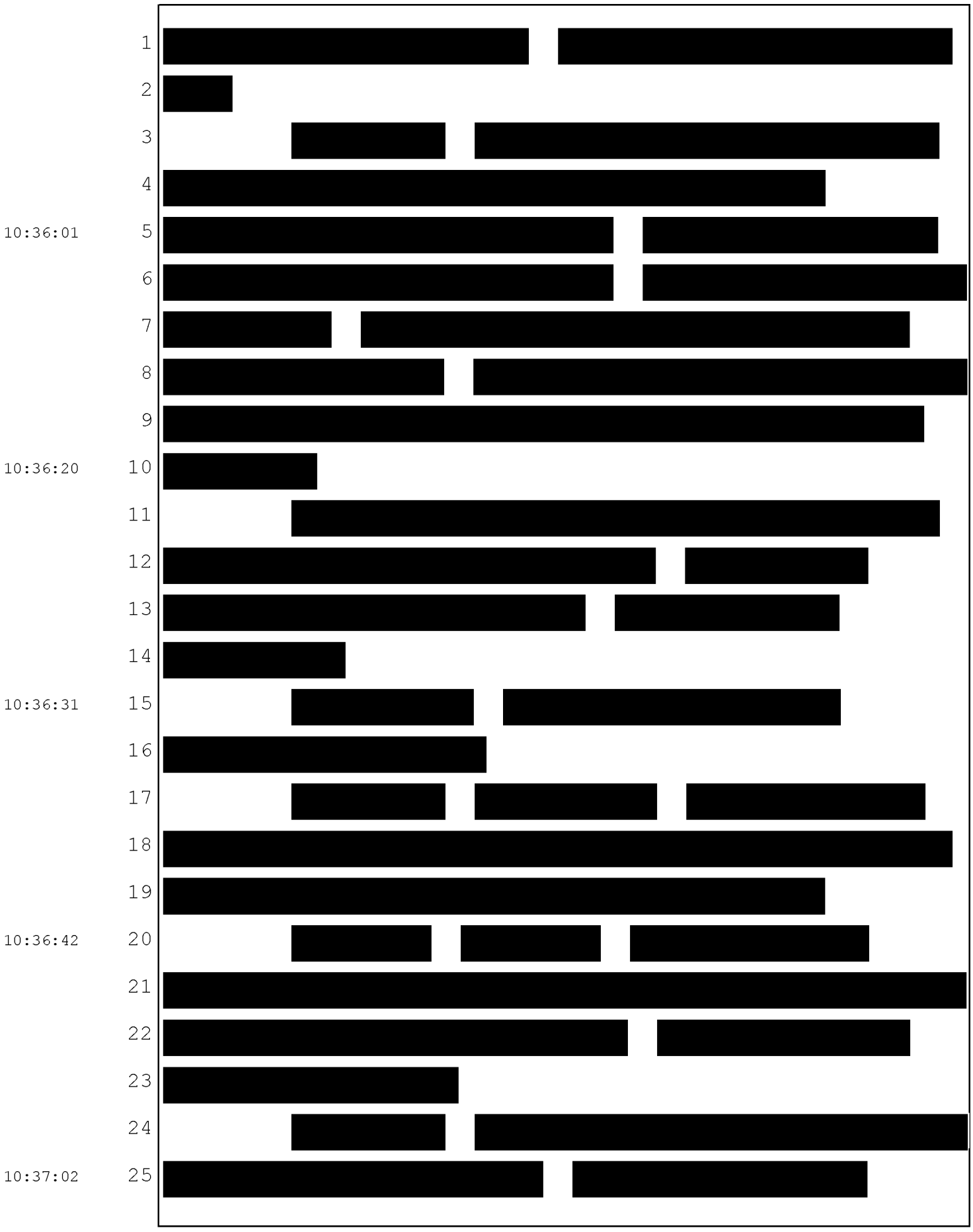
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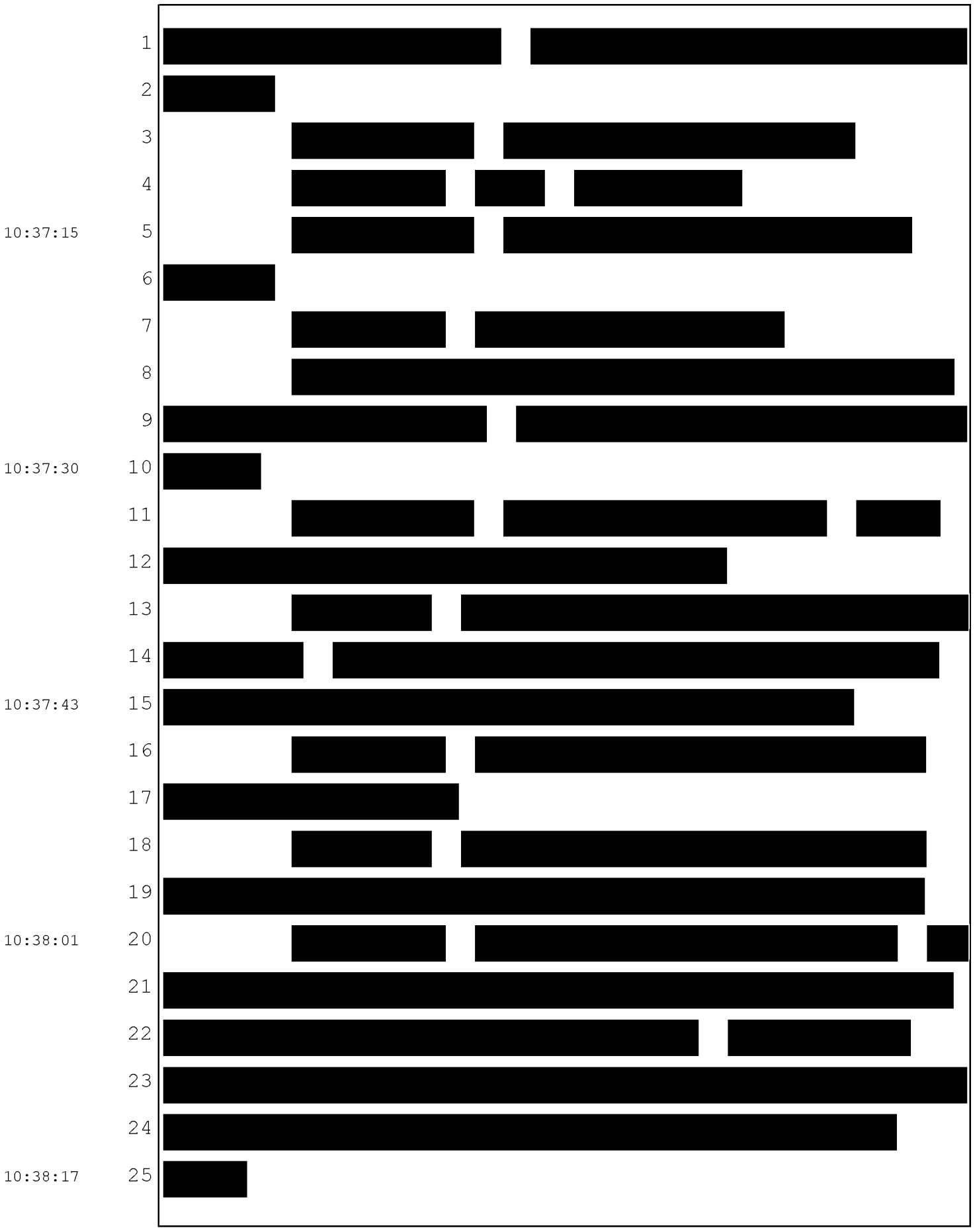
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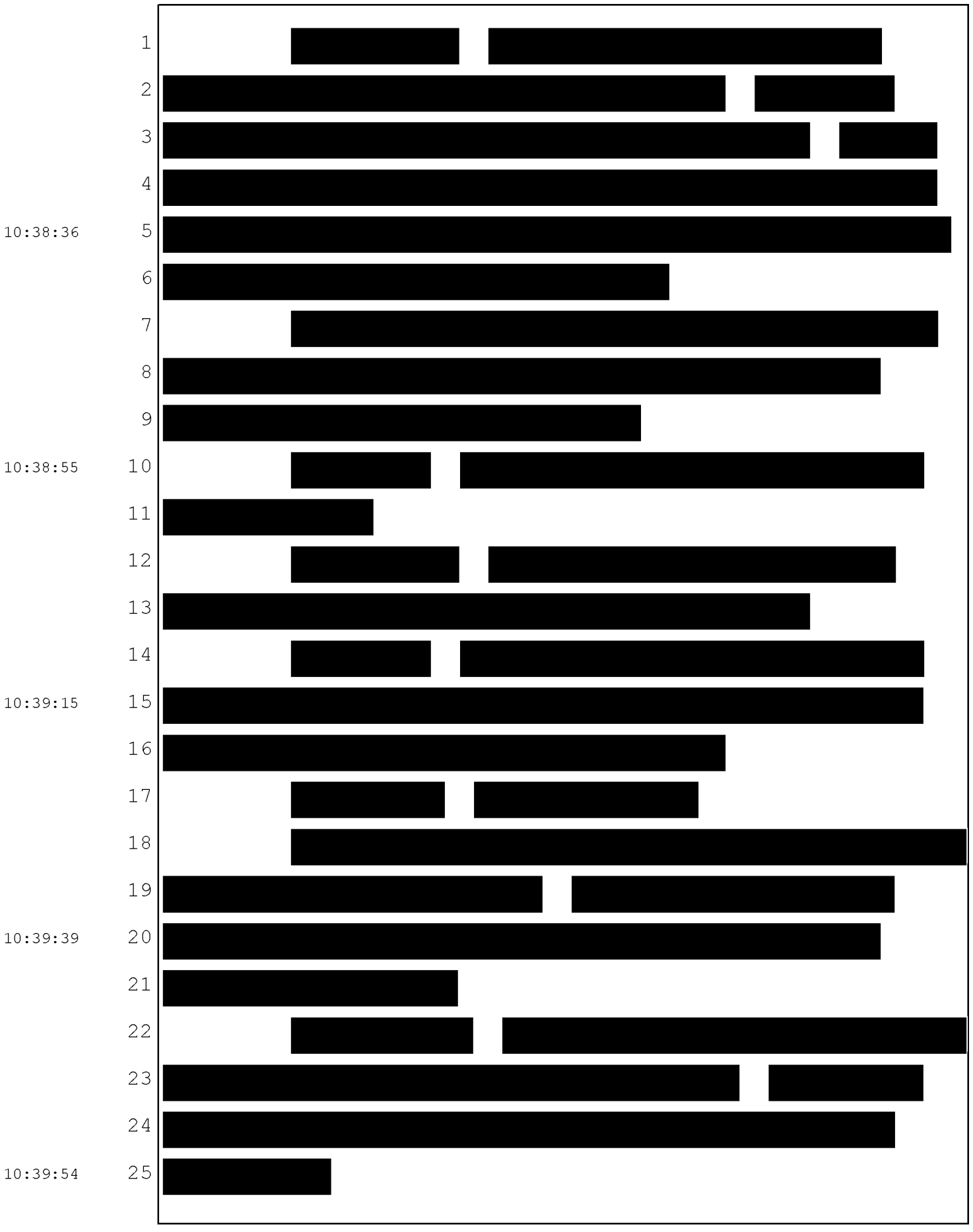
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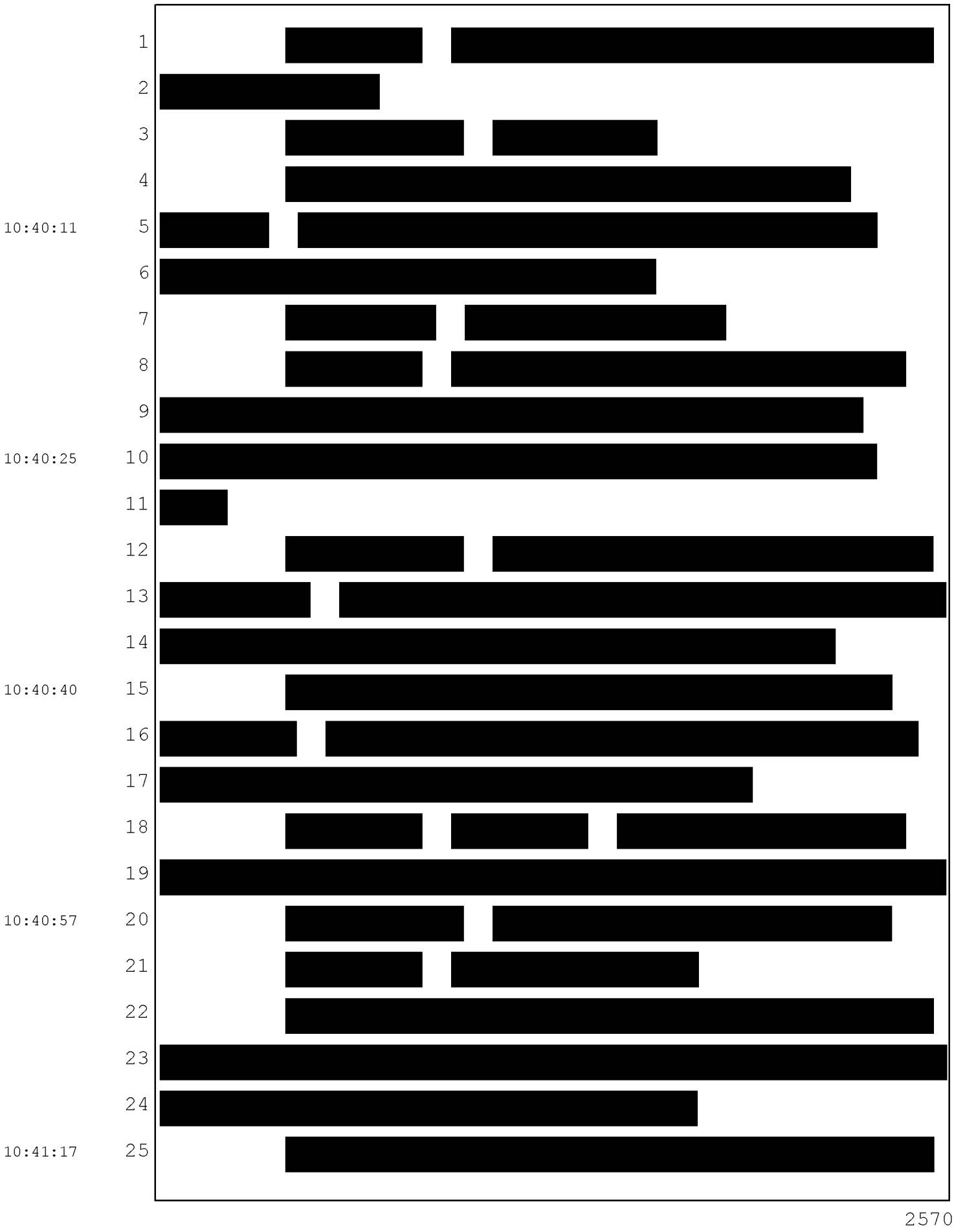
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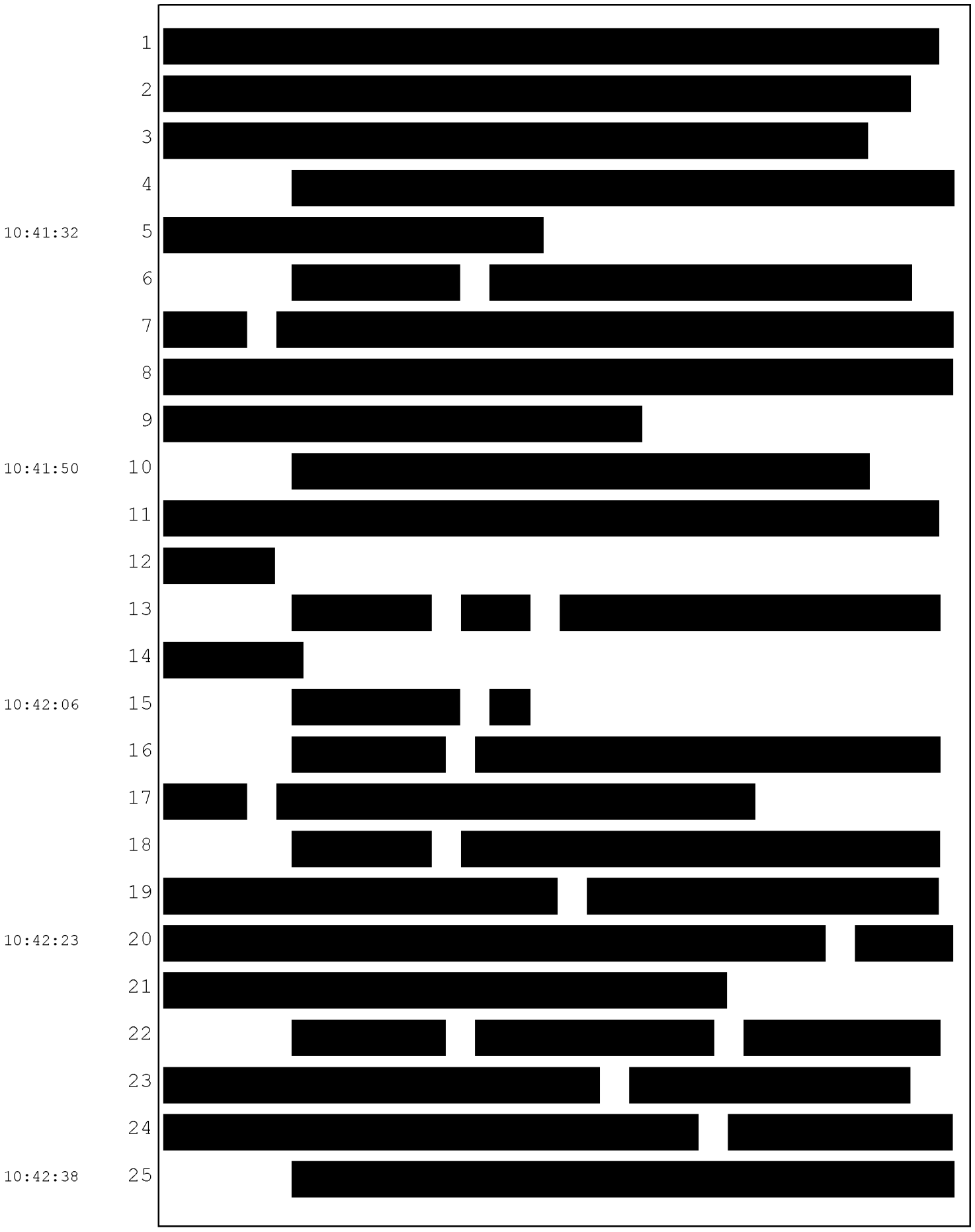
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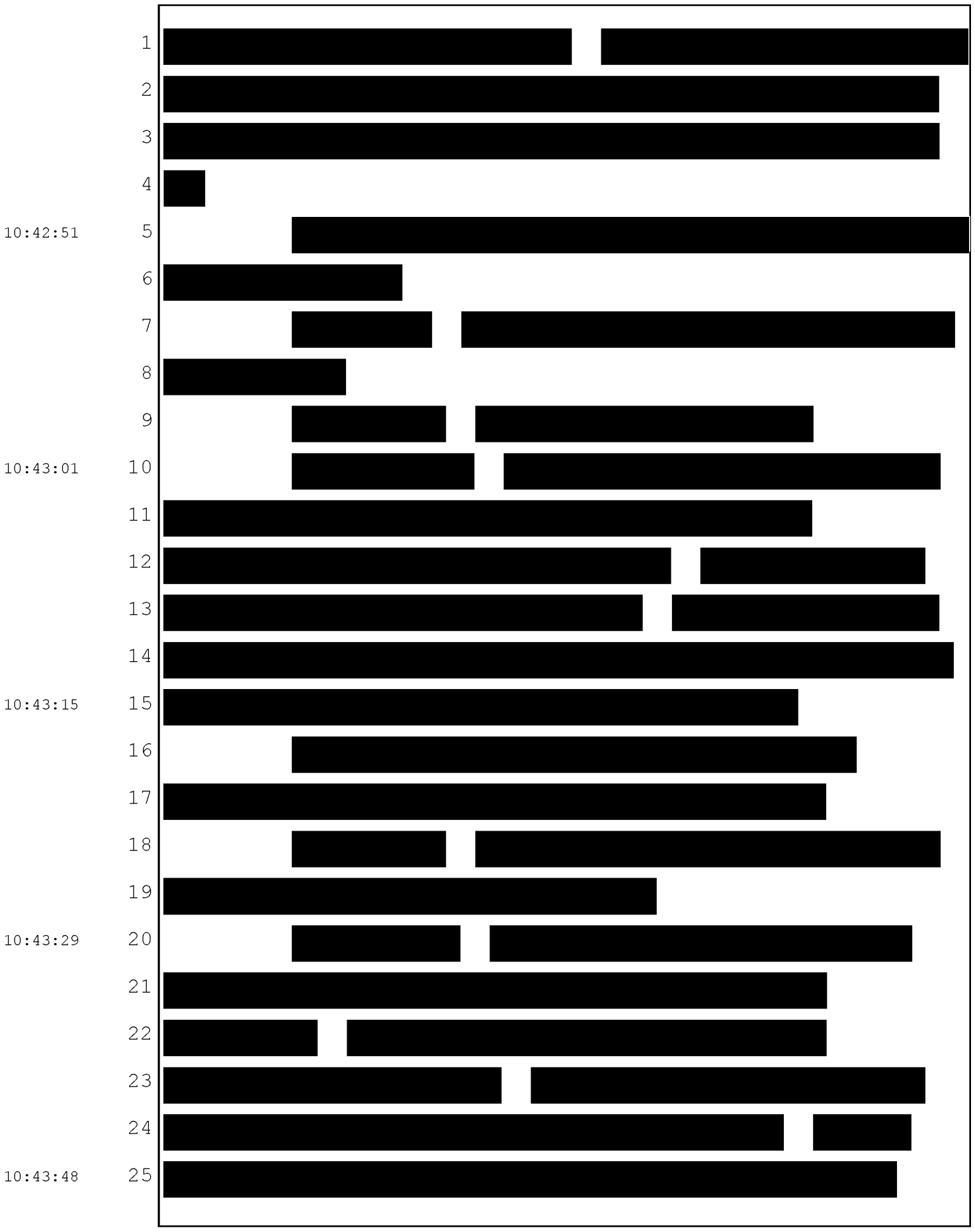
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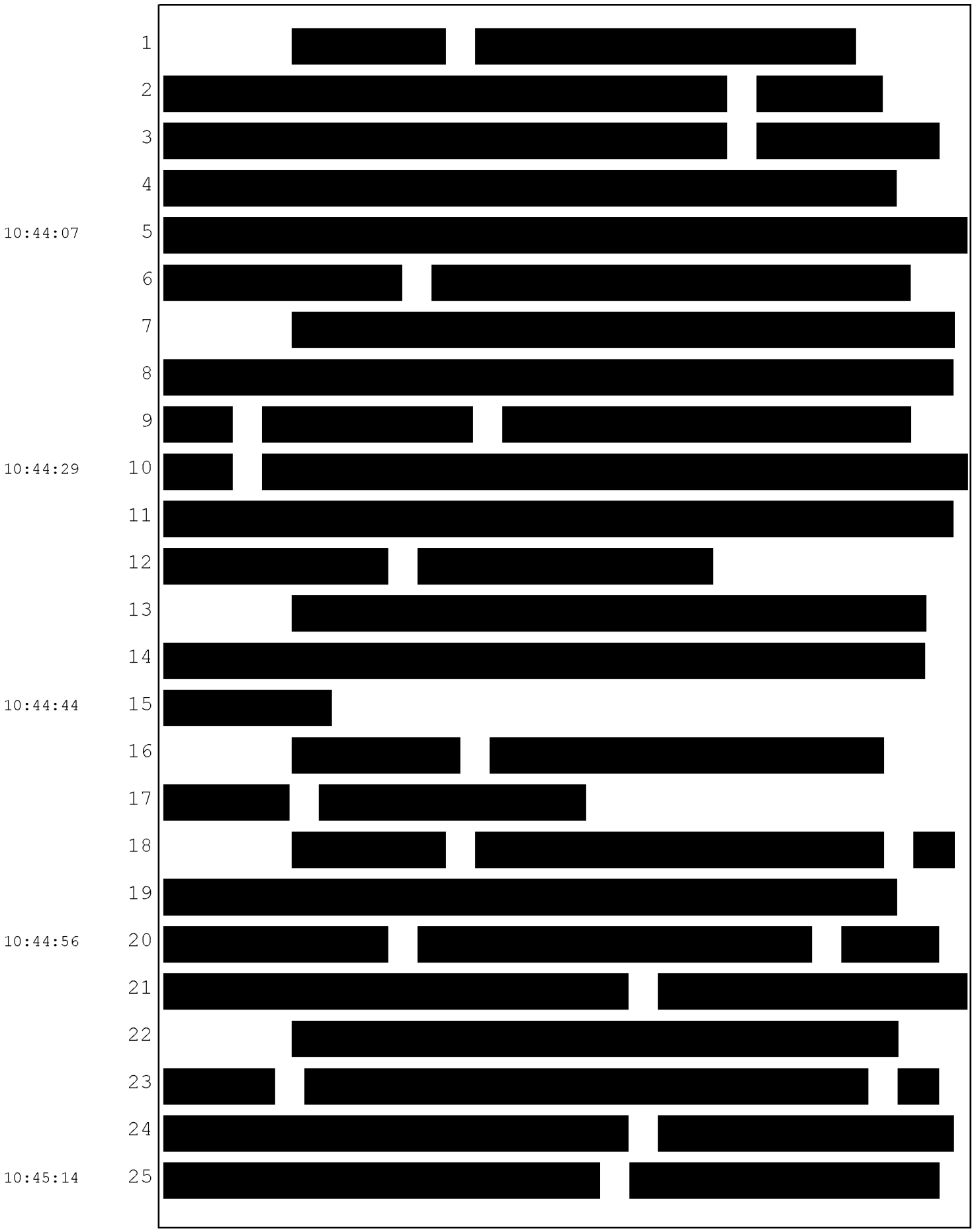
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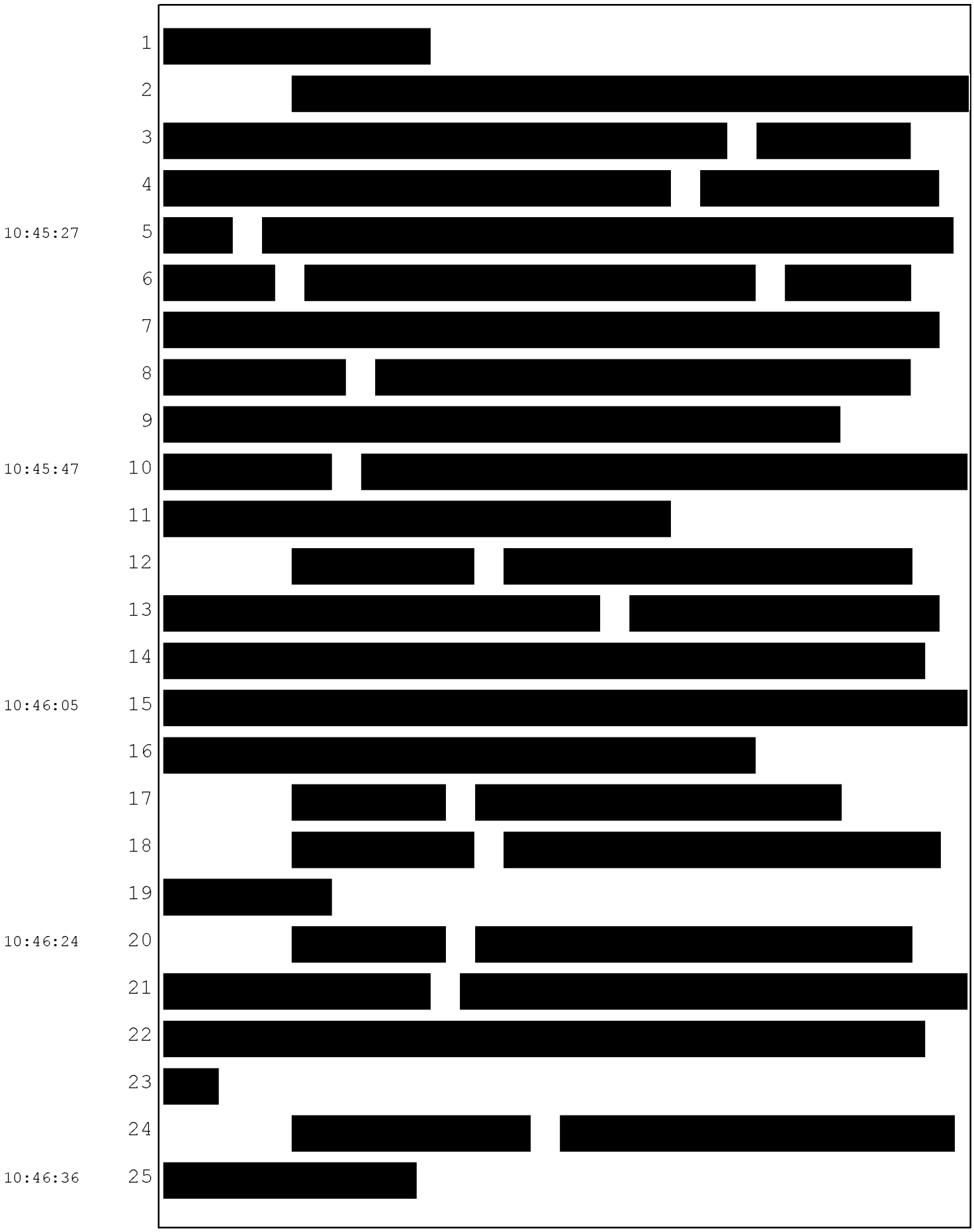
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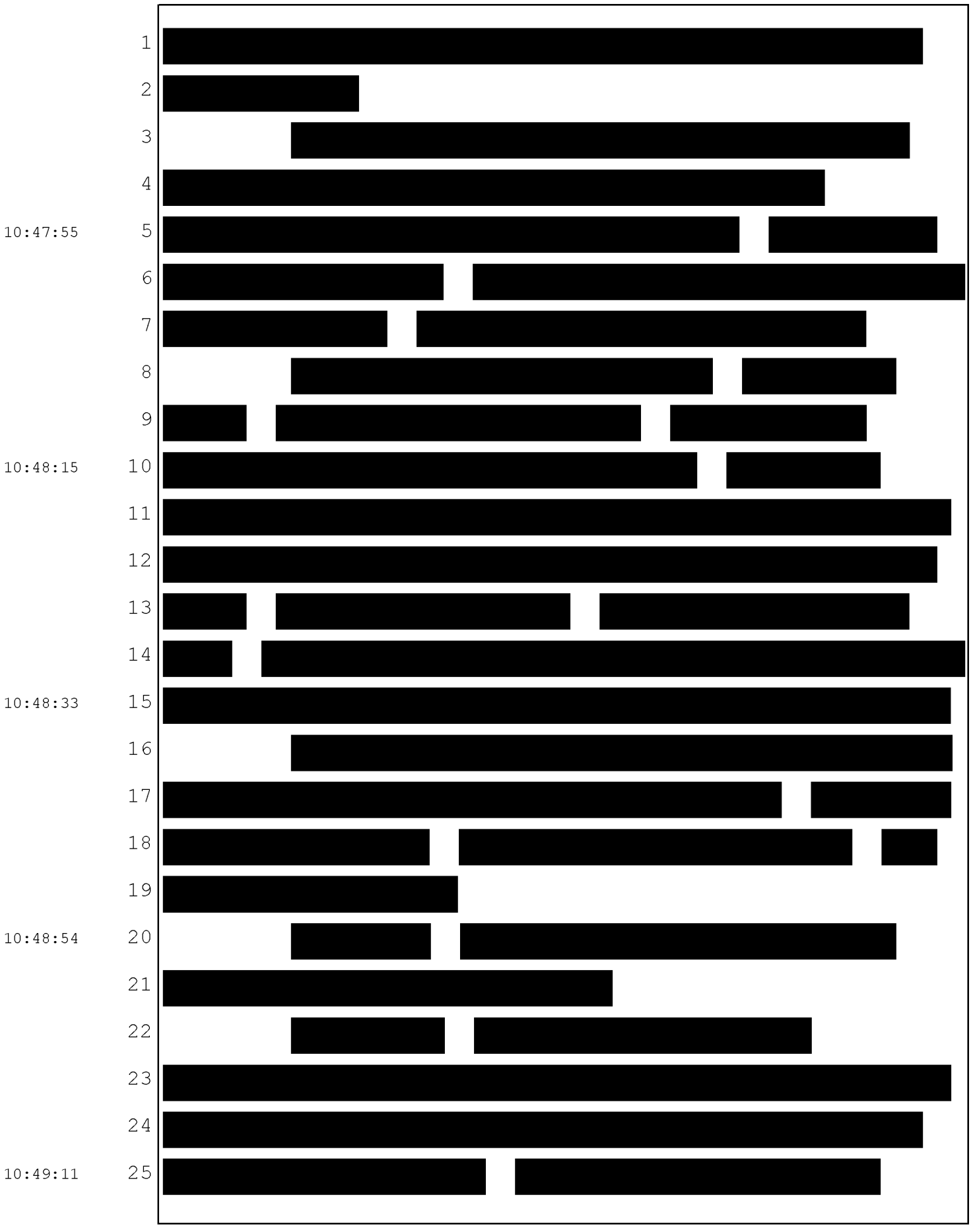
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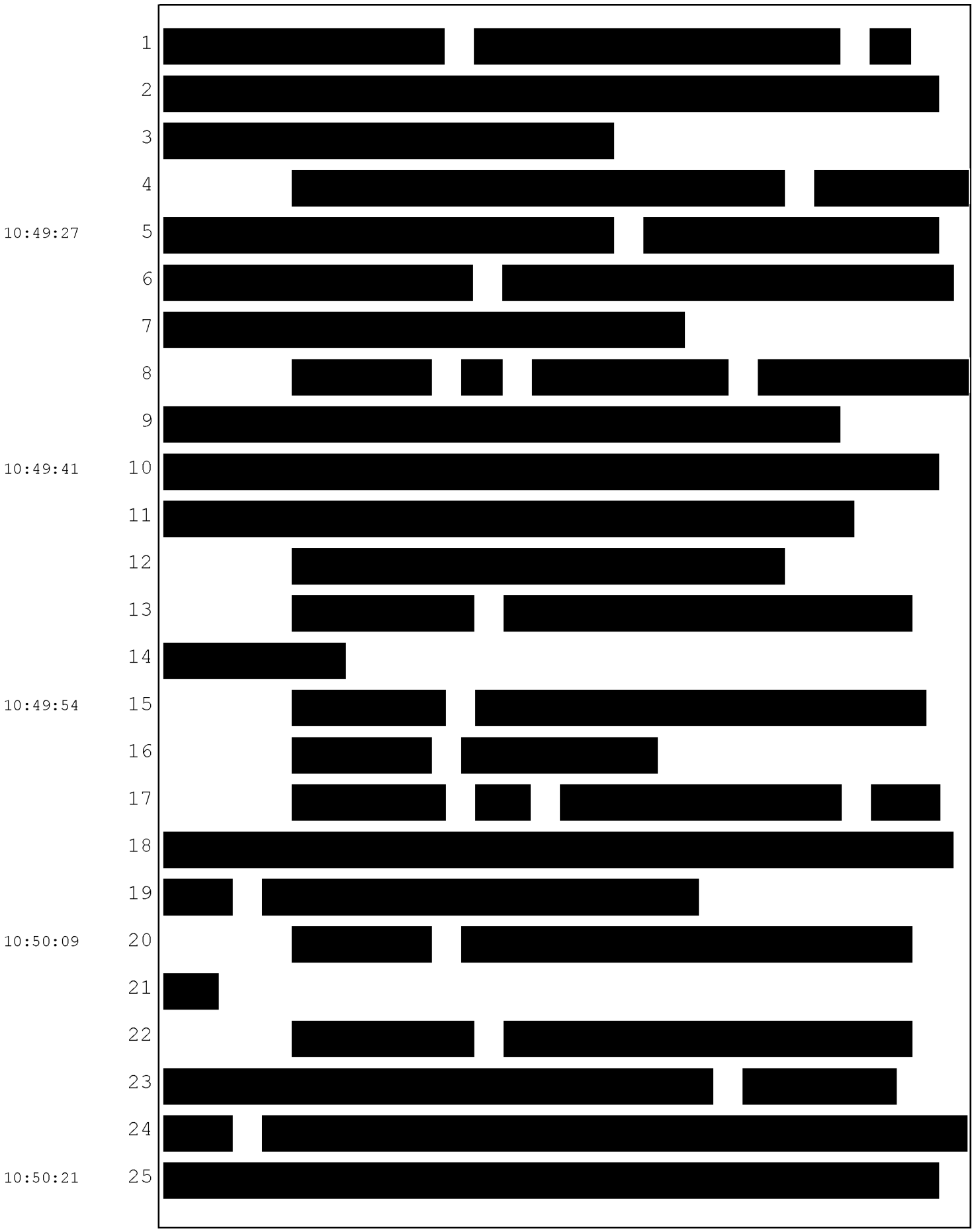
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(End sidebar.)

THE COURT: All right. Mr. Wisner, you may proceed.

10:50:57 MR. WISNER: Thank you, your Honor.

Q. Dr. Neugut, how are you doing?

MR. WISNER: All right. So I'm going to be publishing Exhibit 682. I guess we have an okay on that, your Honor?

10:51:12 THE COURT: Oh, yes. I've overruled the objection.

MR. WISNER: All right.

Q. Doctor, so looking at your screen -- there we go. This is a copy of a document titled "Methodological

10:51:26 Issues Regarding Confounding and Exposure Misclassification in Epidemiological Studies of Occupational Exposure."

Do you see that, Doctor?

A. Yes.

10:51:37 Q. Just before the little break there, we talked

specifically about biases. I want to talk about two of the biases that's actually entitled in this document. One of them is confounding, and one is exposure misclassification.

10:51:52 Do you see that, Doctor?

A. Yes.

Q. All right. What is -- let's start off with the first one.

What is confounding?

10:51:56 A. So confounding is not really a bias. It's a problem that arises in interpretation of an association.

So as I said before, if an epidemiologic study will tell you that there's a statistical association between the exposure and the outcome -- now, let's assume 10:52:21 that that's accurate -- our next job is to say: What is the nature of the association between the two? One is causality, but other things can arise.

So confounding -- you're asking me what confounding is?

10:52:37 Q. That's correct.

A. So confounding is that there is a third factor which explains the association between the two, which is not necessarily causal, so -- and to have confounding, the -- both the exposure and the outcome have to be 10:52:56 associated with this third factor, whatever that might

be.

And it's the third factor that accounts for the association between the two. It's, sort of, an artifactual -- it creates an artifactual association.

10:53:12 A good example might be a -- that you found an association between having yellow fingers and getting lung cancer. So if you did a study, you would find that having yellow fingers is associated with getting lung cancer. But obviously it's not that having yellow

10:53:45 fingers somehow causes you to get lung cancer, but having yellow fingers is associated with smoking. And smoking is associated with getting lung cancer.

So in that association -- again, if you did yellow fingers as the exposure and lung cancer as the

10:54:04 outcome, you would find a statistical association between yellow fingers and lung cancer. But the confounder would be tobacco.

Tobacco is associated with both getting yellow fingers and tobacco is associated with getting lung

10:54:21 cancer. So that's confounding. You're confounded by tobacco, so --

Q. What if we switched that? All right? What if it was the opposite, and we said, "We see an association between smoking and lung cancer" --

10:54:33 A. Right.

Q. -- "but we really think yellow fingers are the confounder"? Would that be a confounder?

A. No.

Q. How do you know it's not a confounder?

10:54:47 A. Well, I would -- so I would say I know it's not a confounder because it's biologically implausible, but -- so that's how I would say it.

But if you wanted to know how you would know from a study, you would -- you would have to

10:55:03 theoretically -- if you wanted to know it from a study, you would probably have to measure it and control for it or do something about it.

Q. But, Doctor, if you control for yellow fingers, right, it would actually eliminate the association with

10:55:16 smoking; right?

A. That's correct. You would do that, that's correct.

Q. So before you control for any confounder, you have to have two parts; right? It has to be associated

10:55:23 with the exposure. So yellow fingers, that's associated with smoking; right?

A. Uh-huh.

Q. But it also has to be connected to the outcome.

A. So if two things are really highly correlated

10:55:33 with each other, it can be hard to sometimes tell them

apart. It's not always easy.

So that's why God gave us brains. We're supposed to actually think when we do studies and use some sense of logic. And this was -- I would say how to
10:55:50 interpret studies, how to interpret associations, how to interpret causality, is not purely a statistical function but we're supposed to use our judgment and our intuition, and our -- and, again, not -- not -- and that's subject to judgment and to thinking and to -- and to the rules of
10:56:16 any other exercise in human behavior of the thought process. So we have to think about it and do other studies and, you know, see how it works.

Q. All right. Let's talk about exposure misclassification. What is that in the context of
10:56:35 epidemiology?

A. So, again, if we go back to our little assessment here, to the degree that you have error in the measurement of either the outcome or the exposure, that's obviously going to cause a problem in terms of how you
10:56:56 assess the risk estimate or how you assess the association between the two.

Most cancer epidemiologic studies, we're pretty good in measuring the outcome. In other words, we know when someone's got cancer and when they don't have
10:57:12 cancer.

I would contrast that, for example, with psychiatric epidemiology. If we're doing studies in depression, when is someone depressed? When is someone not depressed?

10:57:22 If we were studying depression, we might have trouble in measuring with clarity or with validity or with precision the exposure outcome.

But in cancer, we usually know when someone's got cancer and when they don't have cancer. So in most

10:57:43 cases in cancer epidemiology, the outcome is actually measured with a high degree of validity. We know when people have lymphoma, breast cancer, colon cancer, prostate cancer.

The exposure, on the other hand, depending on

10:57:55 the exposure, it may be measured with a high degree of precision, a high degree of validity or not. It depends on how it's measured and what the specific exposure may or may not be.

To the degree that there's exposure

10:58:08 misclassification, that creates an instability in our measurement of the -- of the risk estimate -- of the risk ratio as a relative risk.

Now, this is -- we're talking now about what I would call randomness classification. If this was a

10:58:27 dietary study, for example, and I have to -- how much

broccoli do you eat? No one on earth is going to tell me with precision exactly how much broccoli they eat. So -- so that's going to give me a certain degree of exposure misclassification per force in the study.

10:58:44 And yet we do studies of broccoli consumption and cancer outcomes. So how does that work? The answer is with a certain amount of random error. But when it's assessed in terms of measuring the relative risk estimate, this is random error.

10:59:02 Random error is okay. We can live with random error very well with epidemiology. But what it does is, random error -- in other words, some people measure it a little too high. Some people say, you know, "I eat it three times a week," when they really eat it two times. 10:59:21 Some people, the other way. This is random error.

Random error doesn't give us a biased estimate. It gives us instability in the relative risk estimate. And random error biases towards the null. There's a phrase, it attenuates towards the null, which means it's 10:59:37 a conservative error. That's what I said earlier.

Most errors bias towards the null. We don't want to find a positive finding when there isn't one. We want to find things innocent unless they're guilty.

So what a random error will do is just exposure 10:59:55 misclassification. If we don't measure the exposure

correctly, it will make the risk estimate lower than it might otherwise be. It will bias it -- it will bias it. Will push it towards the null, towards 1. It will make it lower than it really is.

11:00:10 So when we do get the relative risk estimate, that will be an underestimate of truth. So I don't know if I'm saying that well, so everyone understands it.

Q. That's okay.

A. But what -- so random exposure

11:00:23 misclassification, which is really omnipresent, biases towards the null. So unless you have a high-risk ratio -- unless you have a high -- so if your relative risk is 10, like between tobacco and lung cancer -- so heavy smokers have a risk of lung cancer of 10 times

11:00:44 normal, so if you get some exposure misclassification, instead of it being 10, you're going to measure a 9, who cares. It doesn't really make a big difference.

But if you're talking about modest relative risk, like 1.5, like we're talking about in this context,

11:01:01 you may lose that relative risk estimate because of exposure misclassification -- randomness exposure misclassification, and you'll measure a 1 instead of a 1.5. You'll lose it entirely because of exposure misclassification. And you don't need much exposure

11:01:16 misclassification to lose a relevant risk of 1.5.

Q. All right, Doctor. So I'm looking at this article by Dr. Aaron Blair. Who is Dr. Blair?

A. Aaron Blair is the former head of something or other at the National Institute of Environmental Health Sciences. He's a hotshot guy in the -- the environmental and occupational epidemiologic -- I'm one of the leading scientists in this country in this area, and actually, he's a coauthor on many of the papers that are relevant to our discussion today.

11:01:38

11:01:54

Q. He also chaired the IARC program on glyphosate; right?

A. He chaired the -- I don't know if he chaired the whole IARC thing or he -- he certainly chaired the cancer -- yes, he chaired the epidemiology subcommittee. I don't know if he chaired the whole Working Group. But he chaired the cancer -- the epidemiology sub-committee.

11:02:07

Q. So in this study, they state -- they state: "Confounding and exposure misclassification are issues that concern epidemiologists because of their potential to bias results of studies and complicate interpretations."

11:02:27

That's essentially what you just said, right, Doctor?

A. Absolutely.

Q. "In occupational epidemiology, both are

11:02:33

routinely raised to argue that an observed result is either a false positive or a false negative finding. Although it is important to consider the potential for limitations of epidemiological investigations. Judgment
11:02:50 regarding their importance should be based on their actual likelihood of occurrence."

Do you see that, Doctor?

A. Yes. So that's what I was saying before. Judgment is what I was saying before, which is basically
11:03:01 saying that we're supposed to use our brains to think about how to interpret what we see.

MR. WISNER: And, your Honor, I'm just going to ask a few more questions about this, and it will probably be a good time for a break.

11:03:12 THE COURT: Okay.

Q. BY MR. WISNER: All right. Well, so in this study, Dr. Blair and his colleagues, they go systematically through some of the science on confounding and misclassification exposure, and I want to look
11:03:23 through their conclusions.

It says, "Conclusions. We believe that of the two major methodological issues raised in epidemiologic studies of occupational exposures, that is confounding and exposure misclassification, the latter is of far
11:03:37 greater concern."

That's referring to confounding, is that right Doctor? The latter?

A. No, that's referring to exposure misclassification.

11:03:47 Q. Oh, sorry. Yes.

A. I think that's correct, that we don't measure exposure with great precision. When you ask someone how many are -- were you exposed to some exposure -- there's a high error rate in terms of -- it could be a random

11:04:06 error rate, but there's a high error rate, like what I was saying before with broccoli. You just don't get it right. No one does.

Q. They go on to say, "It is rare to find substantial confounding in occupational studies or in

11:04:20 other epidemiologic studies for that matter, even by risk factors that are strongly related to the outcome of interest. On the other hand, exposure misclassification probably occurs in nearly every epidemiological study. For nondifferential misclassification, the type of

11:04:35 misclassification most likely in cohort studies, the direction of bias is largely predictable. That is a bias of relative risks towards the null."

Do you see that?

A. Yes. So that's exactly what I was saying

11:04:50 before, that when you get random error in -- in measuring

the exposure, it will bias the estimate towards the null, towards 1. And by the way, exposure misclassification is going to be -- he says it in cohort studies, but exactly the same phenomenon will occur in case-control studies.

11:05:13 Q. And you would agree, Doctor, with Dr. Blair and his colleagues that the most -- the thing to be most concerned with in evaluating the methodological issues of epidemiology, you're most concerned with exposure misclassification, more so than confounding, although you

11:05:33 are concerned with both?

A. Well, that's the next sentence, if you read on to the next sentence.

Q. Okay. And you agree with that?

A. Yeah.

11:05:39 Q. Okay. Great.

MR. WISNER: Your Honor, it's probably a good time for a break.

THE COURT: All right. Ladies and Gentlemen, we'll take the morning recess now. We'll be in recess

11:05:47 until 11:20 on the clock. Please remember not to discuss the case, and we'll resume again at 11:20. Thank you.

(Sidebar.)

[redacted]

11:06:24 [redacted]


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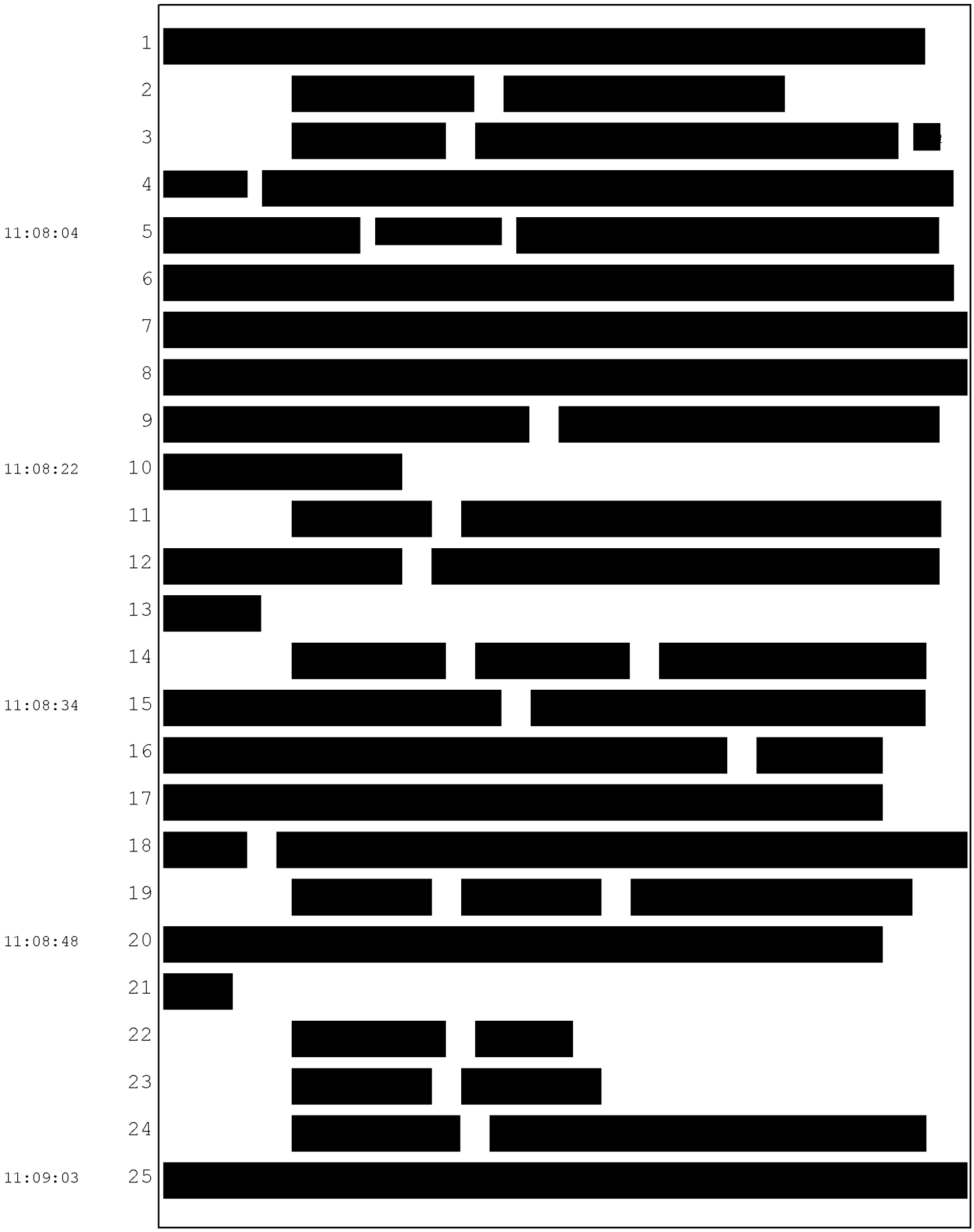
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(Recess.)

THE COURT: Welcome back, Ladies and Gentlemen.

11:21:25 Dr. Neugut remains under oath, and, Mr. Wisner, you may continue.

MR. WISNER: Thank you, your Honor. Permission to publish a slide?

THE COURT: Any objection?

11:21:36 MR. LOMBARDI: No objection, your Honor.

THE COURT: Very well.

Q. BY MR. WISNER: All right, Doctor. We'll continue and see if we can get this worked out as we go.

During opening statements, Mr. Lombardi showed

11:23:06 the jury an image that had a couple of IARC probable carcinogens, one included a -- very hot beverages and nightshift workers. Let's back up.

How can vary hot beverages be a probable carcinogen, Doctor?

11:23:24 A. So in Northern Iran, there's the highest rates

of esophageal cancer in the world by far, and no one knew why, and it turned out in studies that the Iranians there -- I'm not making any political statements. But the Iranians there drink extraordinarily hot tea, and
11:23:52 studies have shown that -- that the tea can -- when it's drunk, can burn the esophagus going down. We're talking about -- we're not talking about the same level of heat that you drink with the Queen of England. We're talking about a level of heat that's greater than, say, 150
11:24:14 degrees, 160 degrees Fahrenheit.

The normal level of tea that, let's say, you and I drink, I guess, or the average American would drink in tea or coffee is about 130, 140 degrees, but they drink it at about 160, 170 degrees, and they drink it right
11:24:31 away after making it. And it shows a high correlation between those who drink this hot level of tea -- and particularly they drink it right after they make it, so they don't wait too long after it's drunk. And they drink tea -- that's their main beverage.

11:24:45 So it scalds -- it scars, basically, their esophagus, and scarring is known to cause squamous cell carcinoma of the esophagus, so that's what IARC is talking about, that kind of -- a high level of esophogeal carcinoma, which applies to hot tea.

11:25:02 The same phenomenon's been described in parts of

China, where they also drink very hot tea. It's not a question in the world about -- no pun intended -- about this being a carcinogenic phenomenon. We're not talking about, again, the tea you get in Starbucks or something like that or that you might make in your home. We're talking about really hot tea.

11:25:23

Q. What about nightshift workers?

A. Nightshift workers are known to be at a high risk for all sorts of medical issues, because they're circadian rhythm has been disrupted. Their hormonal balance of -- when you -- there's twice in a day when your hormones are, sort of, up. I mean, you might recognize it by the fact that you wake up in the morning before your alarm clock does because of your circadian rhythm.

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11:25:57

So if you're going to be, you know, getting up at night and doing nightshift instead of the normal -- like with a rooster every day, that shifts all your hormonal balances, and that relates to getting different kinds of cancer that are -- if you're permanently or usually on nightshift work, then it changes your hormonal balances in such a way as to lead to various cancers. I'm not an expert on that, to be honest, but it's certainly totally accepted within the cancer epidemiology community that this kind of nightshift work causes cancer

11:26:09

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of various sorts.

Q. And here's another slide that was shown to the jury, now that that's working. It says that, "Of the 1,003 classifications made by IARC, only one is probably not carcinogenic."

11:26:47

Is that an accurate reflection of whether or not IARC is, you know, just calling everything cancer?

A. No. That's idiotic.

Q. Why is that?

11:26:56

A. Because that -- that's like proving that something doesn't cause cancer. That's, like -- so in science, we never make that kind -- or very rarely make that kind of absolute definitive statement. The reality is more the next level above, which -- I don't know.

11:27:19

I'll say as I'm sitting here, I'm at a loss of what the next level terminology is.

Q. Unclassifiable, not enough data.

A. Essentially, which would classify as probably 50 percent or more of the chemicals or exposures that

11:27:33

were looked at, which basically says not likely to be associated with cancer, which is more the way we talk in real everyday science and in epidemiology.

So of the thousand-odd chemicals or exposures that have been looked at, most of them are classified as

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probably not -- as not carcinogenic, so that's more

reflective of the reality of the exposures.

Only about 10 to 15 percent of the exposures that IARC has looked at have been classified as 1 -- Level 1 or Level 2A. Glyphosate is 2A -- as Level 1, which is a definite carcinogen, or 2A, which is a probable carcinogen. It's only about 10 to 15 percent of the total are in those that -- are in those two highest categories.

Q. Now, Doctor, please turn in your binder to Exhibit 793.

Are you there, sir?

A. Yes.

Q. This is a letter -- a briefing note from the director of the IARC program in January of 2018.

Do you see that?

A. Yes.

Q. This is something that you reviewed in considering the body of science and information related to IARC?

A. Yes.

Q. I'd like to draw your attention to a few portions of this. First, I'd like to draw your attention to -- I'd like to draw your attention to page 7.

A. Okay.

Q. Okay. And there's a section that reads: "IARC

evaluates only agents that have some evidence of carcinogenicity."

Do you see that? Page 7.

A. Give me a sec.

11:29:45 Q. Middle of the page.

A. Yes.

Q. Okay. I'm just going to read a few of these bullet points and ask you about them. The first one says -- actually, Doctor, just to back up. If you look

11:29:56 to the first page on this, I just want to read what this is.

It says in the first paragraph, "Since the evaluation of glyphosate by the IARC Monograph's program in March 2015, the agency has been subjected to

11:30:12 unprecedented coordinated efforts to undermine the evaluation, the program and the organization. These efforts have deliberately and repeatedly misrepresented the agency's work."

Do you see that?

11:30:23 A. Yes.

Q. So Doctor -- Dr. Wild, who's responding for the director of IARC, is responding to these criticisms of IARC; is that right?

A. Yes.

11:30:32 Q. Seems to be specifically criticisms based on

their classification of glyphosate.

Do you see that?

A. Yes.

Q. All right. So turning back to page 7, the first bullet point under that heading reads: "Some critics say the Monograph's program finds," quote, "everything causes cancer," end quote, "because of nearly 1,000 agents evaluated, only one has been categorized in Group 4, quote, "probably not carcinogenic to humans. The criticism is misleading, because the Monographs do not select at random the agents evaluated for carcinogenicity. Instead, in the interest of efficiency and according to the preamble of -- to the Monographs," quote, "agents are selected for review on the basis of two main criteria, A, there's evidence of human exposure, and, B, there is some evidence or suspicion of carcinogenicity."

Do you see that?

A. Yes.

Q. Now, Doctor, you mentioned earlier that sometimes when you want to look to see if a compound is carcinogenic, IARC just hasn't looked at it. Do you recall that?

A. Yes.

Q. So what is this paragraph telling you about the

way IARC selects chemicals for review?

A. IARC has a process by which it solicits recommendations from general scientific and other communities to decide what agents should be evaluated by IARC. They don't want to waste their resources and time on evaluating everything in the world. So they only review things that have some prior evidence or some suggestion that they may be carcinogenic.

11:31:53

So by the time they're already being evaluated by IARC, they're already a cut above in terms of the likelihood that they're going to be carcinogenic. So if you say that half or not, or -- then you've already taken out like you're -- the pyramidal scale -- you're already in the upper part of the pyramid, in terms of the likelihood that they're going to be carcinogenic.

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And as it says a paragraph or two later, "Despite this careful selection of agents, in reality, around half of the Monograph's evaluations resulted in agents being classified in Group 3" -- that's what I was referring to early -- "not classifiable as to its carcinogenicity in humans."

11:32:46

Which is really where the ones that are not -- where the IARC Working Groups decide that they're really not truly carcinogenic. They fall into -- you know, it would be very difficult to say truly not -- definitely

11:33:03

not carcinogenic or probably not carcinogenic.

Q. Okay. I'll take this off the screen now.

I want to draw your attention to another section that also came up, incidentally, in Mr. Lombardi's opening statement. If you turn to page 8.

11:33:18

A. Page 8 in this document?

Q. Yes, that's right.

It reads: "Monograph evaluations take account of," quote, "real-world exposures by evaluations of epidemiological studies."

11:33:32

Do you see that?

A. Yes.

Q. All right. It reads: "A charge level at the Monographs is that evaluations are divorced from the real world, i.e., are made without taking account of realistic human exposures. However, epidemiological studies are an essential part of Monograph evaluations and by definition deal with people exposed in daily life, including at work. The studies frequently consider the gradient of risk observed with different levels of exposure. One part of the Monograph evaluation is specifically dedicated to describing the circumstances under which human exposure occurs and at what levels."

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And then the last thing it says, "In light of the occurring," quote, "real-world human exposures,

11:34:14

Working Groups synthesize evidence in humans, animals and other model systems in reaching overall conclusions."

Now, Doctor, is it your understanding, as an epidemiologist who relies on IARC routinely in your practice, when IARC classifies something as a probable human carcinogen, does that mean it's not a real-world carcinogen?

11:34:29

A. Of course it's a real-world carcinogen. I mean, it would be nice, speaking as a scientist, to be able to do these studies the same way we do with animals and take 25 or 30 people and put them in a room and give them the maximum tolerated dose and see what happens, but I'm not allowed to do that.

11:34:43

So in essence, we use the -- obviously, the epidemiologic studies are relying on how people are really exposed in day-to-day life. All the studies that we're going to talk about or that we hear are, basically, asking farmers or agricultural workers or whomever how much they've been exposed to. What could be more real life? That's exactly what people are exposed to. That's what epidemiologic studies are.

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11:35:16

Q. Now, if you look at the bottom of this page, the last bullet point, it reads: "In practice, by far the most frequent change in classification after re-evaluation is that the agent goes into a higher group,

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for example, from Group 2A to 1. The fact that most reclassifications move into higher group is an objective indicator that the Monographs do not overstate the strengths of available evidence, but are, in fact, conservative in nature."

11:35:48

Can you explain what you understand that to mean?

A. So it's not uncommon -- again, I don't know the details of this. I'm relying on what they're writing in this document, but generally speaking, over time, of course, more evidence -- whatever classification IARC gives an exposure, things change, and 5, 10 years later, there's more evidence, so IARC may come back and do another Working Group or to -- re-evaluate an exposure.

11:35:59

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And what it's saying is that when that happens, in the vast majority of instances, the reclassification increases the level of carcinogenicity that's assessed. It goes, for example, from a 2A to a 1, from a probable carcinogen to a definite carcinogen. It's very rare that it will go from a probable carcinogen to a less likely -- to a possible carcinogen, down, which tells you that they are very conservative, that they are modest in terms of how they decide on whether -- on the level of carcinogenicity that they assign to a certain exposure, that they usually end up making something even more

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carcinogenic or assign -- reclassify something as even more carcinogenic than they started out with the first time.

Q. Now, Doctor, the reason why IARC classified it in 2A as opposed to 1 was because the epidemiological study was, in IARC's view, limited. Do you recall that?

A. Yes.

Q. Have there been examples in the past where something was originally classified by IARC as 2A because of limited epidemiology, but as time went on, they developed more epidemiology because people are using it in the world and they upped it to 1?

A. So an example of that is formaldehyde. Formaldehyde is a chemical that's used in the occupational setting for a variety of -- in construction, and it's used in the undertaking -- you know, in embalming and things like that.

So that was classified as a 2A originally, and based mainly -- just like glyphosate, based heavily on the toxicological evidence and with limited epidemiology, so it was initially classified as 2A, and then some years later, it had a re-evaluation when there was more epidemiology evidence, more studies had been done, and it got reclassified, and it's now classified as a definite carcinogen, as a Class 1 carcinogen by IARC.

Q. All right. Doctor, let's turn to page 10 of this letter -- of this briefing document.

The third point from the top -- sorry -- the second bullet point. It says, "In fact, identifying
11:38:44 carcinogenic hazards is a crucially important and necessary first step in risk assessment and management. It should be," quote, "a red flag to those charged with protecting Public Health. Revealing that an exposure is a threat or hazard with a Group 1, 2A or 2B
11:39:03 classification should trigger immediate -- either immediate remedial action, for example banned as with asbestos or access to artificial tanning salons for young people, or labeling of carcinogenic hazards or further evaluation of the scale of the risk, risk assessment, in
11:39:22 order to set the levels of exposure to a particular society -- to a particular society is willing -- set the levels of exposure a particular society is willing to accept."

Do you see that, Doctor?

11:39:35 A. Yes, sir.

Q. Can you explain what that -- what you understand that to mean?

A. So what --

MR. LOMBARDI: Objection. Foundation for this
11:39:42 witness to tell what IARC has on its mind. I have no

objection at all to reading through the document and the doctor giving his reactions.

THE COURT: All right. Sustained.

Please ask a different question.

11:39:56 Q. BY MR. WISNER: What is your understanding of IARC's role in epidemiology and cancer research?

A. IARC, as I said at the beginning of my testimony, to me is the number one arbiter in the world of whether something is actually carcinogenic and what 11:40:14 the level of probability is that it is a carcinogen or not. What to do about it or what public policy should be about how to handle that information is for others to decide. I don't think IARC has that as a goal or an intent. It's for other agencies to use the 11:40:35 information -- that information as they see fit.

Q. Now, Doctor, finally, could you just turn to page 3 and going on to page 4. There's a section that specifically relates to Agricultural Health Studies.

Do you see that?

11:40:53 A. Yes.

Q. And my understanding is that the most recent iteration of the AHS as it relates to glyphosate, that was published after the IARC Monograph; correct?

A. Yes.

11:41:03 Q. And in this section, the director is discussing

the impact of that article on the Working Group, to the extent he can.

A. Yes.

Q. Okay. And at the very end of it he quotes testimony from Dr. Blair, who was a chief investigator of the AHS, as well as the chair of the IARC Monograph.

Do you see that?

A. Yes.

Q. And to the best of your understanding, did IARC change its classification following the publication of the Andreotti paper in 2017?

A. No.

Q. Okay. All right. Doctor, I'd like to turn your attention to Exhibit 284 in your binder.

MR. LOMBARDI: Would you repeat the number, please?

MR. WISNER: 284.

MR. LOMBARDI: Thank you.

THE WITNESS: Okay.

Q. BY MR. WISNER: You've got it?

All right. This is a published article titled "IARC Monographs, 40 years of evaluating carcinogenic hazards to humans."

Do you see that?

A. Yes.

Q. And this was published, it appears, in 2014; right -- sorry -- June of 2015?

A. 2015.

Q. That's right. So this is after the glyphosate listing by IARC in March of 2015?

11:42:32

A. Yes.

MR. WISNER: Permission to publish, your Honor?

THE COURT: Any objection?

MR. LOMBARDI: No objection, your Honor.

11:42:40

THE COURT: Very well. You may proceed.

Q. BY MR. WISNER: So this is the article, Doctor. And what they're doing in this article, and correct me if I'm wrong, is they're reviewing has IARC been correct and effective in the last 40 years since its existence; is that right?

11:42:56

A. Yes.

Q. All right. And if we look here at the list of authors, there are over 120 authors on this publication; isn't that true?

11:43:07

A. I didn't count them, but I'll take your word for it.

Q. Okay. And this would be consistent with your testimony earlier that IARC is widely respected within the academic community; right?

11:43:18

A. Reading through the list, I see many well-known,

very famous cancer epidemiologists who are highly respected in the world.

Q. I see one of them is -- Dr. Aaron Blair is actually the second author; right?

11:43:31 A. Yes.

Q. Okay. Now, I don't want to spend too much time on this. We have limited time with you today, so I just want to highlight the conclusion. It should be on your screen, Doctor. Can you read it?

A. Yes.

Q. It says, "Disagreement with the conclusions in an IARC Monograph for an individual agent is not evidence for a failed or biased approach. Some disagreement about the carcinogenic hazard of important agents seems

11:43:59 inherent to the scientific enterprise and is unavoidable at early stages of hazard identification where IARC usually operates."

What does that mean? The stages of early hazard identification, what does that mean?

11:44:20 A. I guess it's saying when it's being first evaluated, where there's not necessarily complete data.

Q. Okay. And then it says, "Because the violations are not and should not be static, it is difficult to see how such assessments could be addressed any differently.

11:44:36 Substances now universally recognized as human

carcinogens, for example tobacco or asbestos, at one time went through a quite lengthy period of contentious debate. Any process in theory can be improved with fair and conservative criticism. Appropriate reviews may take
11:44:56 place from time to time, and we would support continued review and improvement of the IARC processes. However, as a group of international scientists, we have looked carefully at the recent charges of flaws and bias in the hazard evaluations by IARC Working Groups, and we have
11:45:13 concluded that the recent criticisms are unfair and unconstructive."

Do you see that?

A. Yes.

Q. Do you agree with that?

A. Yes.

Q. If you had been asked as one of these 125 other scientists who signed this article, would you have?

A. I'm upset that I wasn't.

Q. I -- just for my own education, Doctor, have you
11:45:28 ever seen 125 scientists agree on anything?

A. I suppose, but not too often.

Q. Okay. All right. Let's move off of IARC. Let's go into some epidemiology here. I don't want to spend too much time on it. We had a chance to talk to
11:45:49 Dr. Portier a bit about some of the studies, but I would

like to discuss a Forest plot. I understand you had one prepared for your testimony today; is that right?

A. Yes.

MR. WISNER: Okay. Permission to publish?

11:46:00 THE COURT: Any objection?

MR. LOMBARDI: No objection, your Honor.

THE COURT: Very well.

Q. BY MR. WISNER: All right. Doctor, what is this?

11:46:27 A. So this is what is referred to as a Forest plot. A Forest has nothing to do with trees. It's actually named after someone called Forest, but -- and what it's showing is -- actually, can I just for a moment --

THE COURT: Yes, you may step down.

THE WITNESS: It won't take me more than a moment.

Q. BY MR. WISNER: Do you need a marker?

A. Not necessary. So one -- so these are what are called relative risks or risk ratios. One means that 11:47:11 everything is equal, what I alluded to earlier, that there's no association between the exposure and the outcome. So it -- there's no exposure between -- no association between the exposure and the outcome, and you measure the association. You get a value of 1. So the 11:47:28 closer you are to 1, it means there's no association, so

1 means there's no association.

And these are one, two, three, four, five -- six studies. These are all case-control studies, like we alluded to earlier. Case-control, meaning that you started with people who had lymphoma, and controls, people who did not have the lymphoma.

Q. Let me just interrupt. De Roos 2005, that's actually a cohort study; right?

A. Oh, I'm sorry. Yes.

And then looked at their exposure to glyphosate or their association with glyphosate. And if your risk ratio is above 1, that means that you have an increased risk -- an association of having an association between the exposure and the outcome. If it's below 1, it means that there's a protective effect, that, in fact, glyphosate -- theoretically, that glyphosate would actually protect you from lymphoma, if you're -- if you measure 0.8 or 0.7. It means you have a 30-percent lower risk of having lymphoma if you were exposed to glyphosate.

Theoretically, if everything was truly random, according to the null hypothesis, like we said -- like things should be, if it was truly random between glyphosate and non-Hodgkin's lymphoma, then these studies should be randomly distributed around 1. Half should be

above. Half should be below. That's what random means. Half should be above and half below.

And if you look, all of them are above 1. All of them. That's a phenomenon referred to in causal epidemiology as consistency. They're consistently elevated above 1. Whatever flaws, problems, issues we're all going to raise about these studies, one or the other, no studies are perfect, whatever things each study does, no study is identical. One does something. One -- each study does something differently. Each study -- but all the circumstances under which these six studies -- some of them control for different things, some of them are done in different populations. Some of them in Scandinavia. Some of them are in America. Some of them in Canada. Some of them are with farmers. Some of them are not.

But all of them are consistently above 1, and that's none random. And here -- this is what's called a meta-analysis, (inaudible) but it's not --

Q. Meta RR.

A. Meta RR.

So you get what's called a meta RR. So what happens here is a study was done by some scientists, by Cheng and Delzell. And what they did was they put together the risk estimates. Basically, they combined

these six studies together to get a combined risk ratio so we would be able to see what the combined outcome was from the six case-control -- six studies. They're not all case-control, and see what the combined effect was of
11:50:47 all of them.

And this is the combined effect, and the outcome is that the risk ratio was 1.3, meaning that there was a 30-percent increased risk of non-Hodgkin's lymphoma in the context of glyphosate exposure, with a significant
11:51:10 evaluation -- with a significant -- statistically significant 95-percent confidence interval, as it's called, so that's the combined effect of all.

Why do you need to combine them to get them? So the point is that all of them, or almost all of them,
11:51:27 cross the 1 line, which means that none of them are statistically significant on their own. We like to have statistical significance if you can. We don't have that in most of the studies. Why don't we have statistical significance in most of the studies? That's because of
11:51:45 what I alluded to earlier, which is we're dealing with a very uncommon outcome and a very uncommon exposure.

So to have a statistically significant outcome in any individual study is extremely difficult, but if you combine them -- and again, the fact that they're all
11:51:59 consistently positive together makes them -- leads to a

statistically significant positive exposure.

The truth is that in the De Roos 1.6, they're using -- they took for this particular.

Q. You can cross it out and put in the right number.

11:52:23

A. They took a different -- the paper contains a whole slew of analyses. And probably they took a conservative estimate from it. They were trying to be conservative, like we always try to be, but they probably should have taken one where the risk estimate was .1. And so if they had taken that, it probably -- it would have been statistically significant -- De Roos would have been statistically significant on its own, and you probably would have had a somewhat higher risk ratio here.

11:52:38

11:52:53

Again, I'm not going to argue whether it's 1.3 or 1.4, et cetera. That's not really the issue. The point that we should walk away with is that overall, there's a statistically significant increased risk in the 1.3, 1.4, possibly 1.5, range. And that's basically what the case control studies are showing us.

11:53:06

Q. Thank you, doctor. Great. That was easy.

I want to point out a few things the jury should see, because I think it's a little different than -- they saw a plot summary earlier with Dr. Portier. I want to,

11:53:29

kind of, explore some of the differences.

A. Did Dr. Portier show this?

Q. No. He showed a different chart. This is from a publication; is that right?

11:53:39 A. Uh-huh. Yeah, uh-huh.

Q. Okay. So that's why this 2.1 is -- it's not something you would have selected. You would have selected the 2.1 if you were making your own chart.

A. Uh-huh.

11:53:49 Q. Okay. And a couple things: I notice the scale here is a little different than the one we saw before. This has .1 to 1, and then 1 and 10.

A. Uh-huh. That's what's called a log scale.

Q. Okay. So if you were not to do a log scale, you know, .1 would be right there and 10 would be -- who knows where; right?

11:54:03

A. Yeah, uh-huh.

Q. Okay. This is designed to, sort of, push everything towards the 1; is that right?

11:54:10 A. That's correct.

Q. Okay. And notwithstanding that design, you still see a fairly consistent either at 1 or to the right of one; is that right?

A. It would look more dramatic if you did not

11:54:21 have -- if you used a straightforward numerical scale.

Like a regular integer scale.

Q. Okay. Great.

Now, one of the things that I also want to bring up here is you mention one study here, the De Roos 2005 study.

11:54:39

A. Yes.

Q. Actually, before we go there, these are the most conservative numbers from all of those studies; right?

A. That's correct.

11:54:45

Q. So there are -- for example, like in Eriksson, there's actually a 2.-something number that's statistically significant, that's not adjusted for other pesticides.

A. That's correct. So that would show in the dose response relationships, yes.

11:54:59

Q. Yeah. And that's where we're going next.

So you're actually using the most conservative numbers here; right?

A. Again, that's what these -- the authors who made the Forest plot in the meta-analysis did. And I think that's -- again, we should be conservative and try not to, you know, overstate reality.

11:55:08

Q. Now, I understand this is just never ever; is that right?

A. Yes.

11:55:25

Q. So this is not reflecting if someone used it for, like, more than two days a year?

A. Correct.

Q. Now, some of the authors did actually look at, sort of, dosing or exposure effects; right?

A. Yes. Yes.

Q. And I believe it was in McDuffie and Eriksson; right?

A. Yes.

Q. And in both of those studies, both of the elevated exposure groups have statistically significant rates of NHL?

A. So while these overall analyses are, as I say, not statistically significant for the most part, that's because you're also including people who ever -- someone used glyphosate for two days, you know, and that was it, they're still included here as a positive glyphosate person, but they're -- obviously that level of exposure is not gonna make any significant contribution to the risk of getting lymphoma.

On the other hand, if you start to look at dose response of people who are really significantly exposed to glyphosate, got exposed in a more dramatic way, for longer periods of time, for higher doses, they're going to have a significantly higher risk.

And when you look in some of the papers, you see that dose response, and then you see much more significant levels of risk ratios that are statistically significant.

11:56:44 Q. All right. So we have on here De Roos 2005. And that's from the agricultural --

A. AHS study.

Q. Okay. Great.

They've heard a little bit about the AHS study, 11:56:55 but I want to hear from an epidemiologist. They published the new results, the new ones, last year; right?

A. Yes.

Q. You've reviewed them?

11:57:05 A. Yes.

Q. You considered it?

A. Yes.

Q. Do you think that that study is a reliable study?

11:57:10 A. No.

Q. Why?

A. For a few reasons. So -- so first, you have to appreciate that why -- what changed. So first of all, the level of use of glyphosate between the initial 11:57:29 exposure from -- let's say from 1993 to 1996, the people

were initially recruited in the 1993 to 1996 time frame.

What happened subsequent to 1993 to 1996 was that there was a dramatic rise in the use of glyphosate for various reasons, so that whatever was assessed in terms of the initial exposure of glyphosate really became almost useless information. I think we have a poster --

11:57:59

Q. A demonstrative. Do you want to show the jury that?

A. If we can.

Q. Yeah. Absolutely.

11:58:11

MR. WISNER: Permission to publish 1032?

THE COURT: Any objection?

MR. LOMBARDI: No objection, your Honor.

THE COURT: Very well.

THE WITNESS: So, again, we're talking now about a cohort study. So we're talking about people who were exposed -- who were recruited in 19 -- 50,000 people roughly. 50-something-thousand people who were exposed between -- who were recruited between 1993 and 1996 or so. And they were each given a questionnaire and asked about their usage of glyphosate and other herbicides.

11:58:19

11:58:40

And then what happened is -- you can see here, after 1996, there was a huge rise, almost tenfold, in the use of glyphosate.

11:58:57

So, basically, if you relied on the level of

glyphosate exposure that you had collected on the initial questionnaire back in 1993, '94, when you questioned them, it's totally useless. I mean, because their use of glyphosate has dramatically increased in between.

11:59:16 This doesn't usually happen with most exposures. If I ask someone, "How much do you smoke," and they said, "I smoke two packs a day," and then five years later you ask them how much they smoke, they're still smoking two packs a day. But here the glyphosate exposure went up

11:59:34 dramatically.

So basically what it says is you can't rely on the baseline questionnaire that was done in 1993, 1994, 1995, because the glyphosate exposure has dramatically changed. So, okay, that's no one's fault.

11:59:48 So the investigator said, "Okay. Here's what we have to do: We have to go back and reinterview everybody," which is exactly what they did.

So they went back and tried to reinterview everybody --

12:00:00 Q. BY MR. WISNER: And that was actually in 2001 and 2005; right?

A. Right. So around 2005, they went back and tried to reinterview the 50,000-odd, not odd, but, you know, 50,000-ish people who were in the study.

12:00:13 So now they run into what's a problem in cohort

studies. What's a problem in cohort studies is you've got to try to find them and get them to -- to go back and do the questionnaire again.

Well, I don't have to tell you, if someone
12:00:28 called -- this was by telephone. I don't know what you do. When someone calls me on the telephone and wants to interview me, I hang up. Or I don't answer the phone in the first place.

So what happened was they called the 50,000
12:00:43 people. Again, it's a little more than 50,000, but I'm just using rough numbers for our discussion. When they called the 50,000 people, they got a cooperation rate, if you want to call it that, a follow-up rate, of 62 percent. So 38 percent of the people that they
12:01:00 called -- I don't think anybody here will be surprised by that -- 38 percent of the people did not respond. Or they did not get follow-up from 38 percent of the cohorts.

Now they don't have information on 38 percent of
12:01:12 the cohorts in terms of their subsequent follow-up in terms of glyphosate.

Q. So, Doctor, let me just interrupt you before we get an objection.

A. Yes.

12:01:20 Q. For example, let's say they had been -- between

1992 and 1997, they'd never used glyphosate.

A. Right.

Q. Okay? And then in 2000, they start using it; right?

12:01:32 A. Yeah. And if you didn't interview them, you wouldn't know anything about it.

Q. And then let's say it's even worse. let's say -- 2000, they started using it for a couple of years. They died in 2004 from non-Hodgkin's lymphoma.

12:01:45 Okay?

A. Yeah.

Q. Let's say that happens. Then when they called, they obviously couldn't answer the questionnaire.

A. Because they'd be dead.

12:01:54 Q. Yeah. So that person, for purposes of the study, then, would be considered unexposed, and the cancer would then be assigned to the unexposed group?

A. Well, it's not clear what would happen. No, that person would have been imputed as being unexposed.

12:02:09 Yes, that's what you said.

Q. Yeah, that's what I meant.

A. Uh-huh.

Q. And that's exactly misclassification of exposure. That's the thing we were talking about with

12:02:18 Dr. Blair.

A. That's correct.

Q. So what happened?

A. So what happened is -- so, correctly, what they did was -- then you have several problems. So, again, this would not be a problem -- this would not be a problem if the risk ratio was high. If the relative risk was 10, like the tobacco and lung cancer -- again, these are errors that, kind of, would have been made -- instead of a relative risk of 10, maybe we'd have a relevant risk of 9 or a relative risk of 8 and -- who cares.

If we're talking about a relative risk of 1.5, then these kinds of errors are enormous and -- or potentially enormous. And so they -- they get thrown out in the wash, so to speak, so --

Q. Let's stop right there.

A. And there are several errors here.

Q. Let's stop right there.

MR. WISNER: Your Honor, it's probably a good time to take a break, so we can get a time check and make sure we don't run into the buzzer.

THE COURT: Okay. Great.

All right, Ladies and Gentlemen. Then we'll -- we'll break now for the lunch recess. Please remember: Do not discuss the case, do not do any research. And we'll see you again at 1:30. Thank you.

(Jury leaves courtroom.)

(Time Noted: 12:03 p.m.)

REPORTER'S CERTIFICATE

I certify that the proceedings in the within-titled cause were taken at the time and place herein named; that the proceedings were reported by me, a duly Certified Shorthand Reporter of the State of California authorized to administer oaths and affirmations, and said proceedings were thereafter transcribed into typewriting.

I further certify that I am not of counsel or Attorney for either or any of the parties to said Proceedings, not in any way interested in the outcome of the cause named in said proceedings.

IN WITNESS WHEREOF, I have hereunto set my hand: July 18th, 2018.

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