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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:

LESLIE ROCKWOOD ROSAS, RPR, CSR 3462

Job No. 2965316A

Pages 2512 - 2625

1 APPEARANCES:

2

3 FOR THE PLAINTIFF:

4 R. BRENT WISNER, ESQ.

5 BAUM, HEDLUND, ARISTEI, GOLDMAN PC

6 12100 Wilshire Boulevard, Suite 950

7 Los Angeles, California 90025

8 310-207-3233

9

10 DAVID DICKENS, ESQ.

11 THE MILLER FIRM, LLC

12 108 Railroad Avenue

13 Orange, Virginia 22960

14 540-672-4224

15

16 FOR THE DEFENDANT:

17 SANDRA A. EDWARDS, ESQ.

18 FARELLA BRAUN + MARTEL LLP

19 235 Montgomery Street

20 San Francisco, California 94104

21 415-954-4400

22

23

24

25

1 APPEARANCES (Continued):

2

3 FOR THE DEFENDANT:

4 GEORGE C. LOMBARDI, ESQ.

5 JAMES M. HILMERT, ESQ.

6 WINSTON & STRAWN LLP

7 35 West Wacker Drive

8 Chicago, Illinois 60601

9 312-558-5969

10

11 KIRBY T. GRIFFIS, ESQ.

12 HOLLINGSWORTH LLP

13 1350 I Street, N.W.

14 Washington, D.C. 20005

15 202-898-5800

16

17

18

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EXHIBITS ADMITTED

(None.)

1 Wednesday, July 18, 2018

2 9:25 a.m.

3 Volume 12

4 Morning Session

5 San Francisco, California

6 Department 504

7 Judge Suzanne Ramos Bolanos

8  
9 PROCEEDINGS

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11 [REDACTED] [REDACTED]

12 [REDACTED] [REDACTED]

13 [REDACTED] [REDACTED]

14 [REDACTED] [REDACTED]

15 09:25:13 [REDACTED] [REDACTED]

16 [REDACTED] [REDACTED]

17 [REDACTED] [REDACTED]

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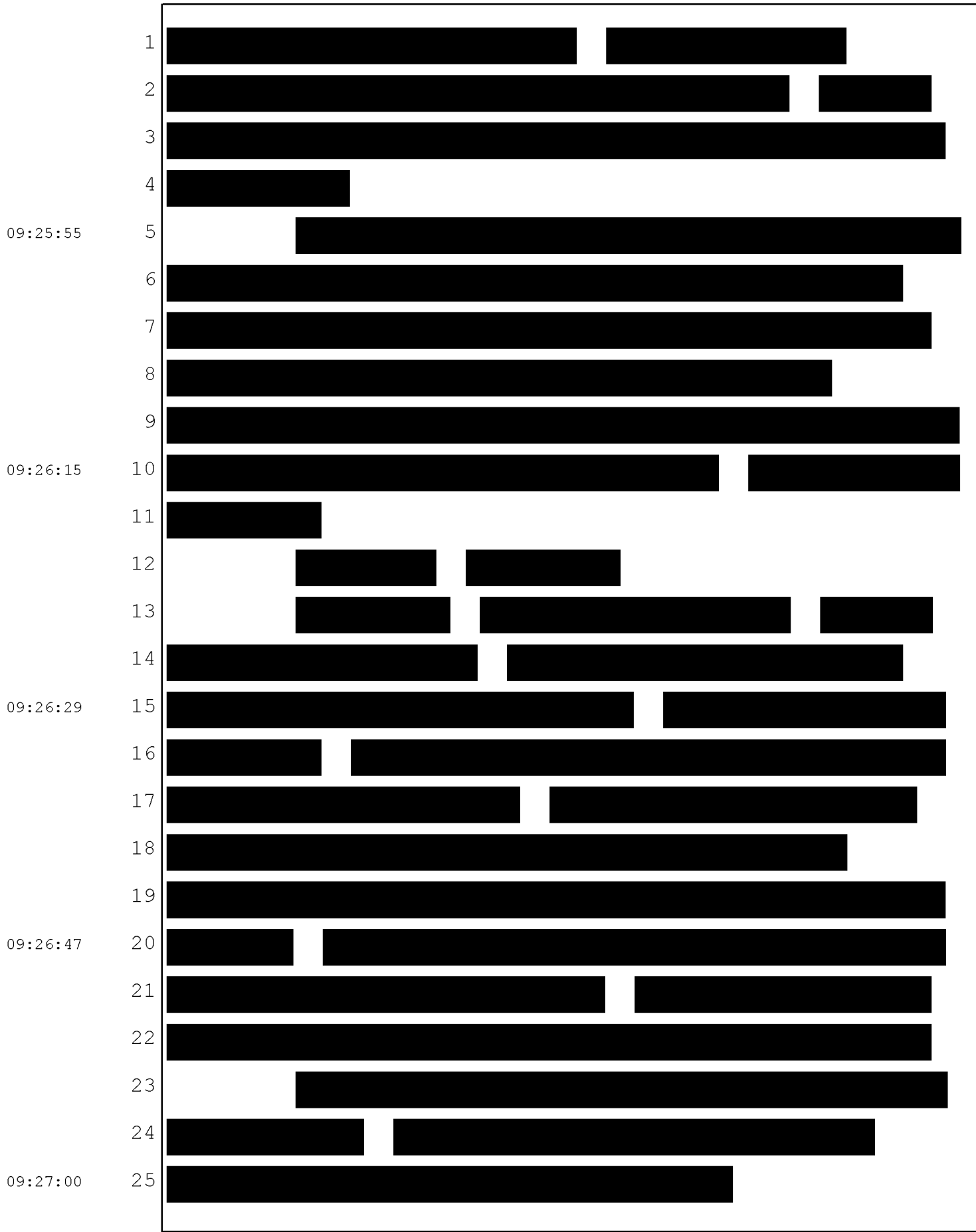
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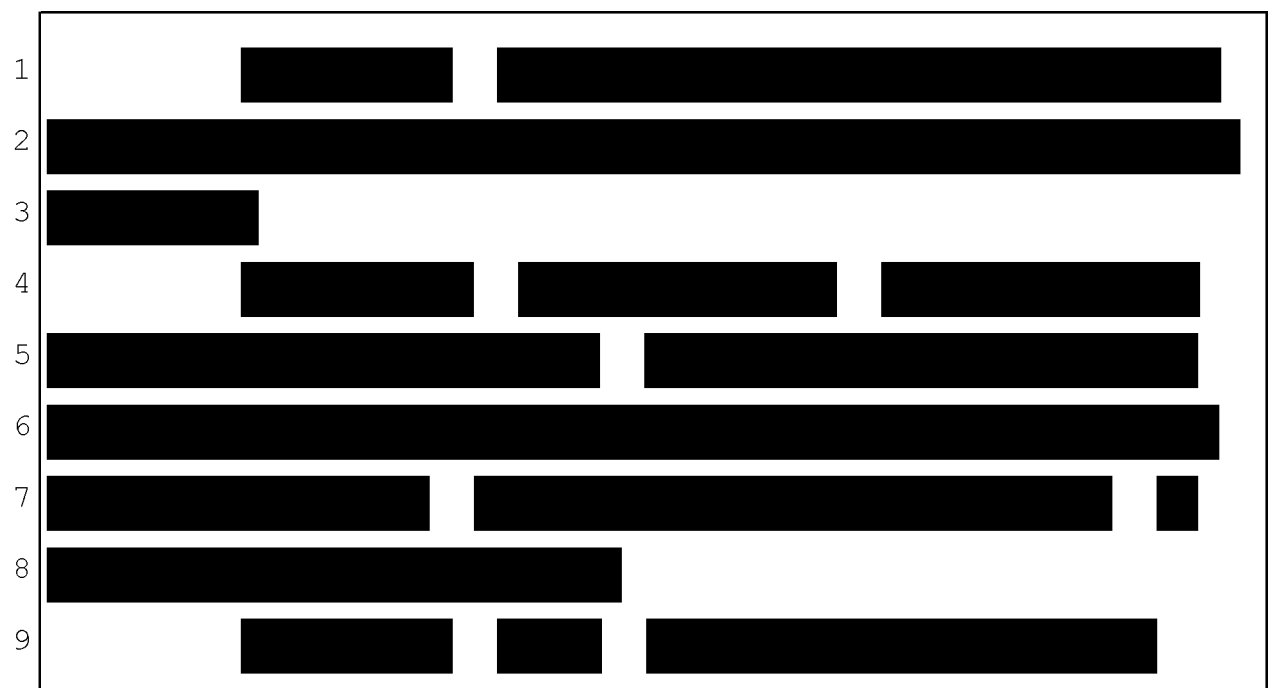
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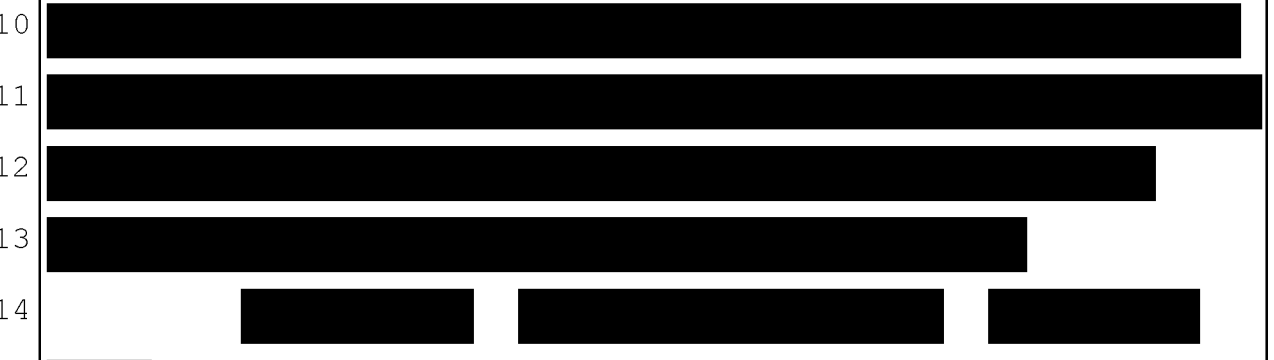
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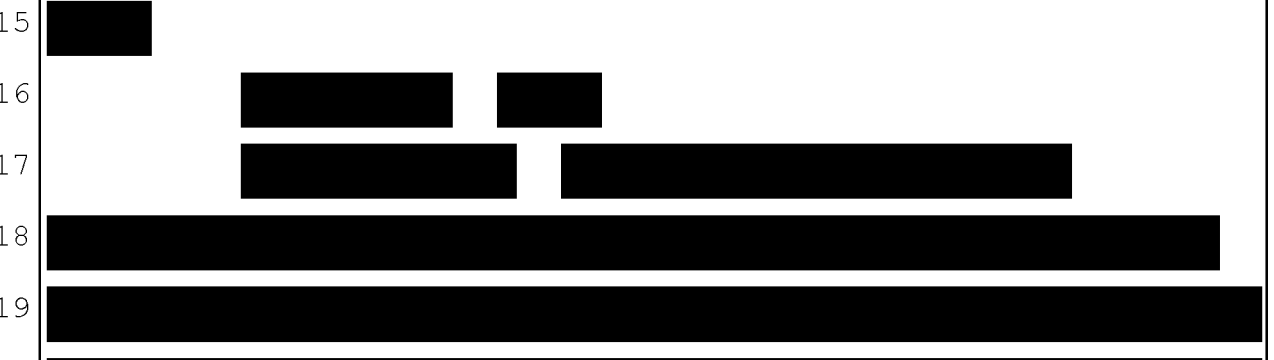
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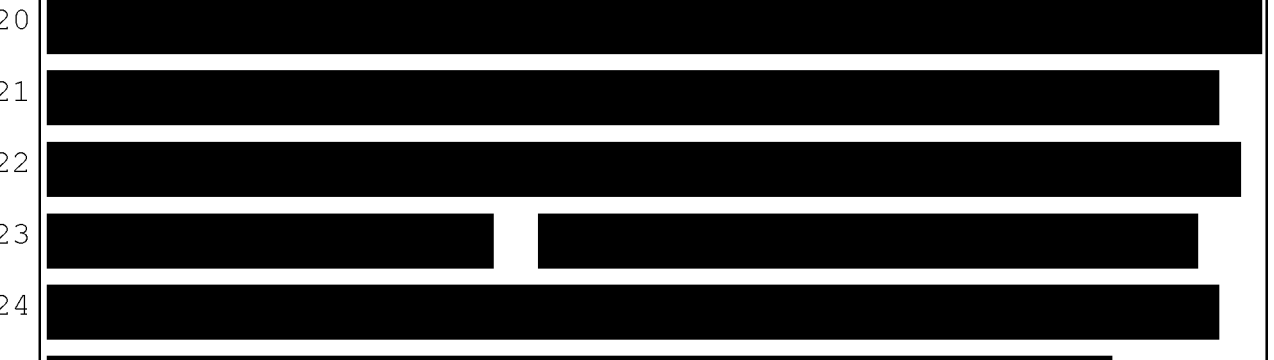
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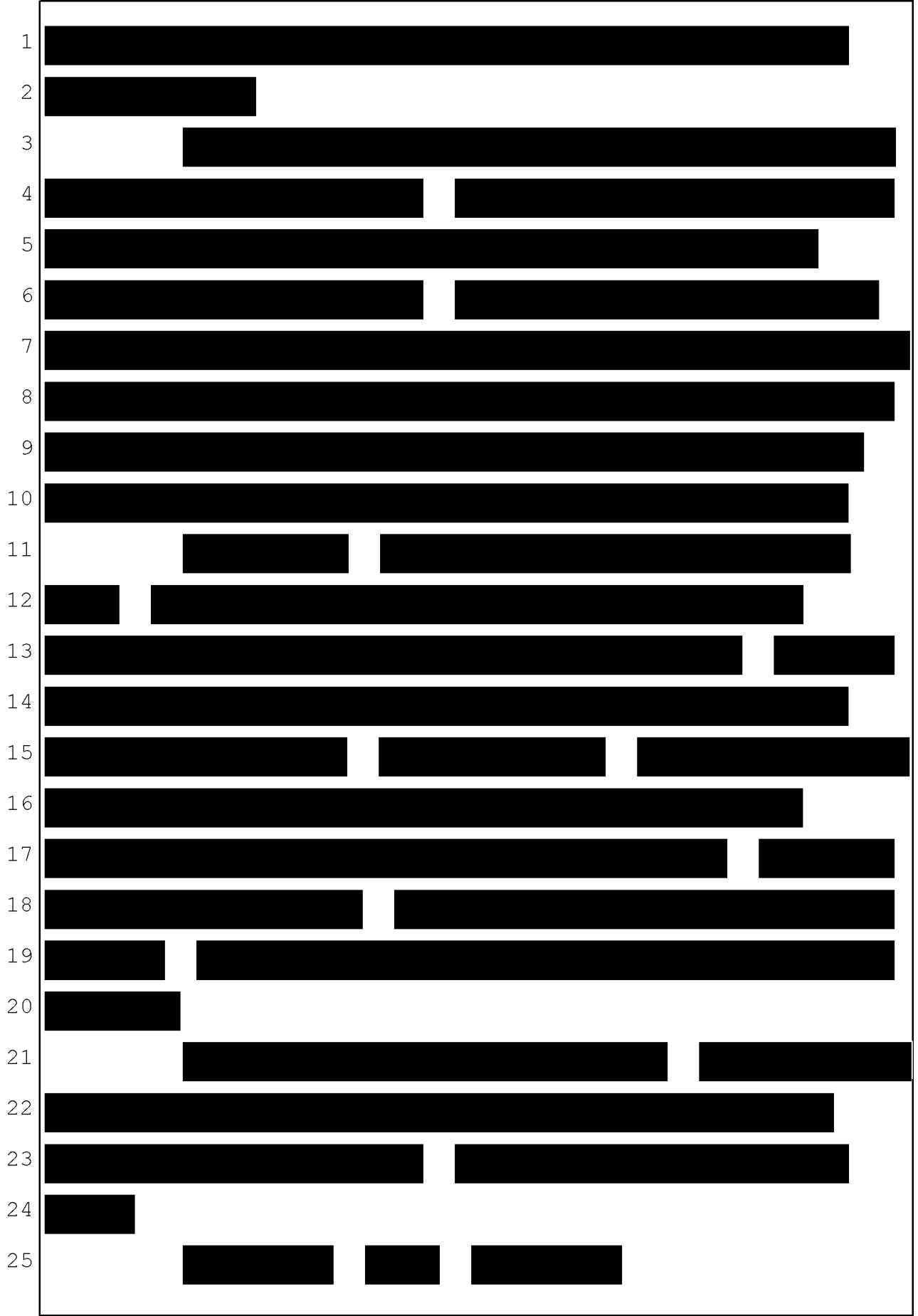
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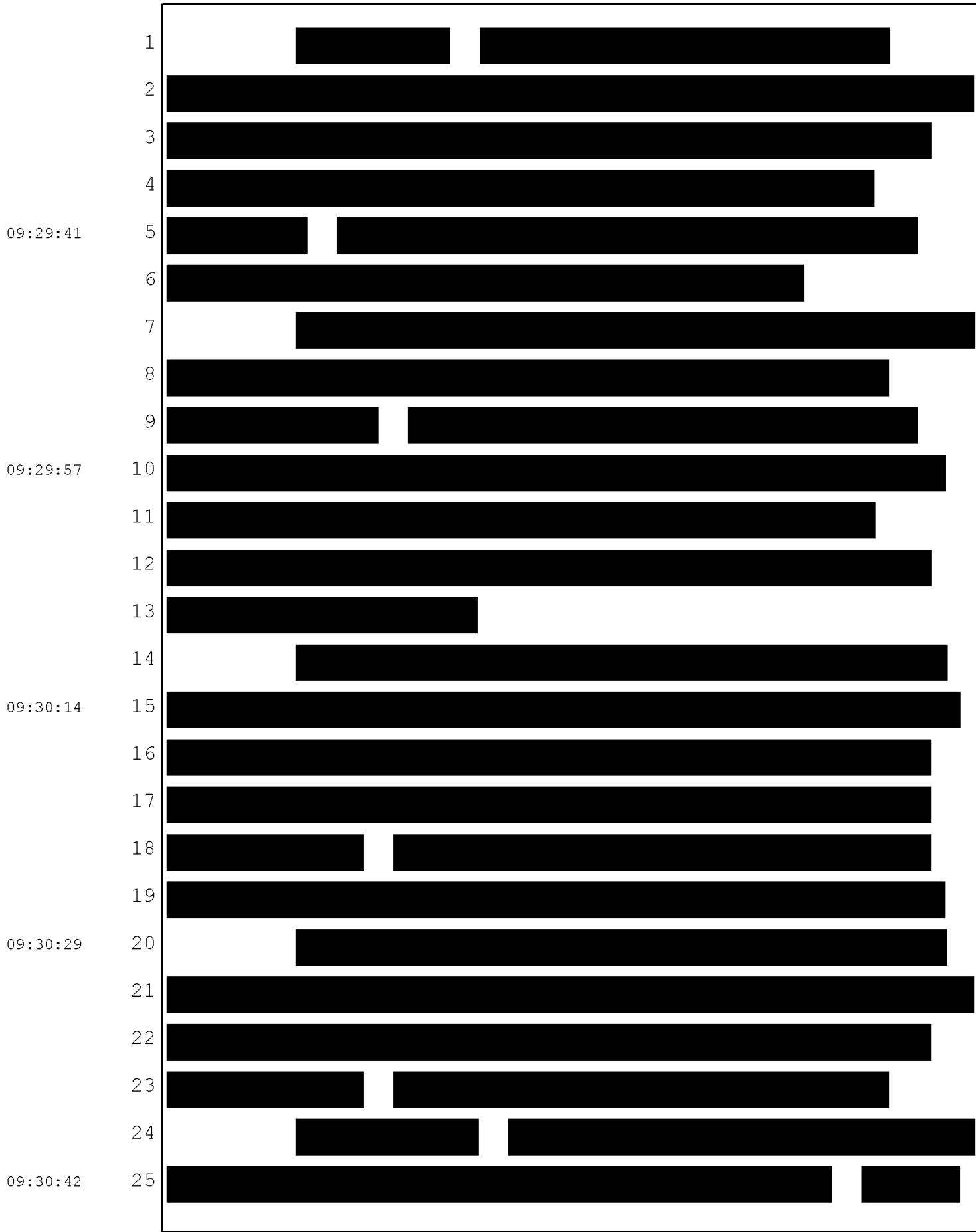
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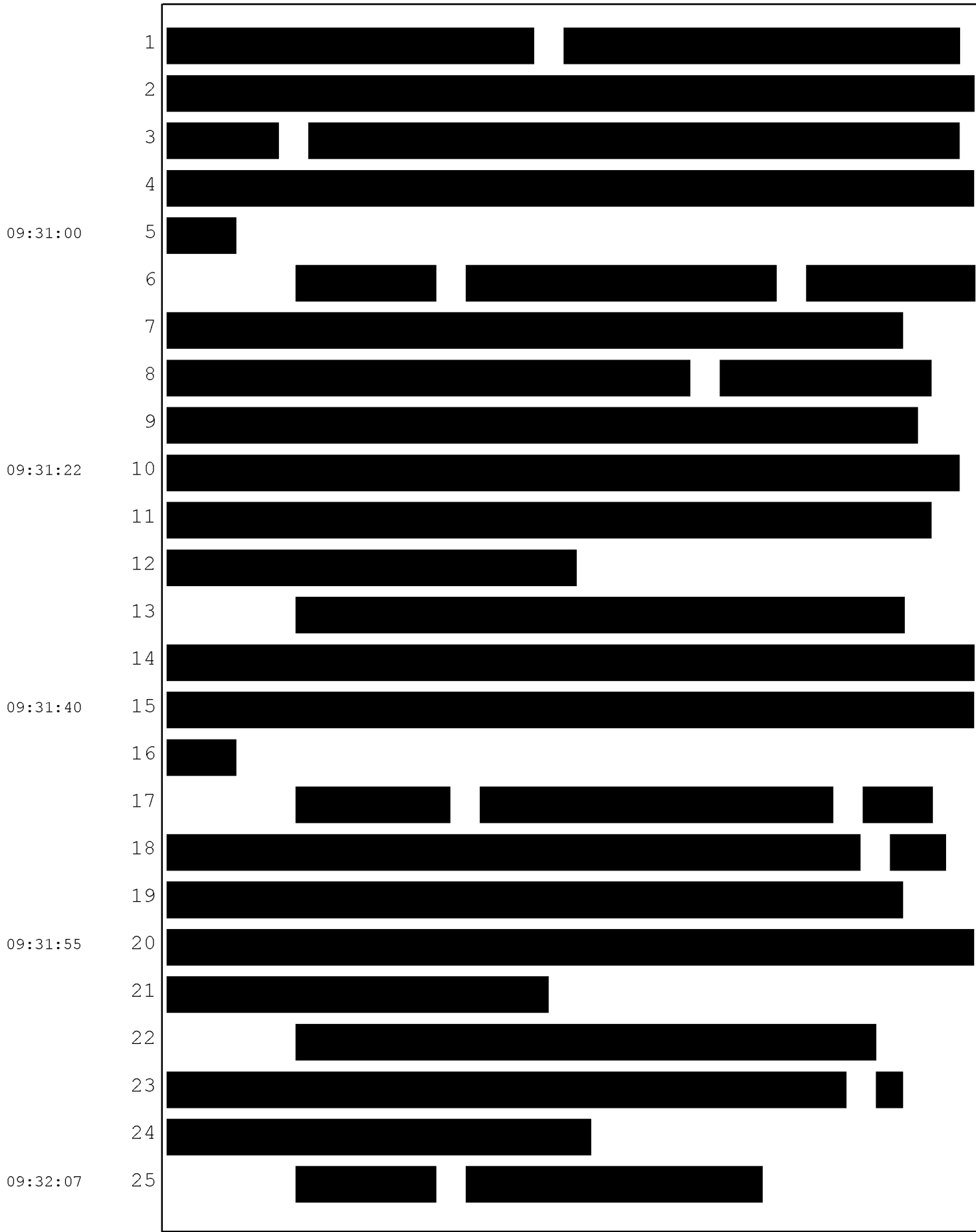
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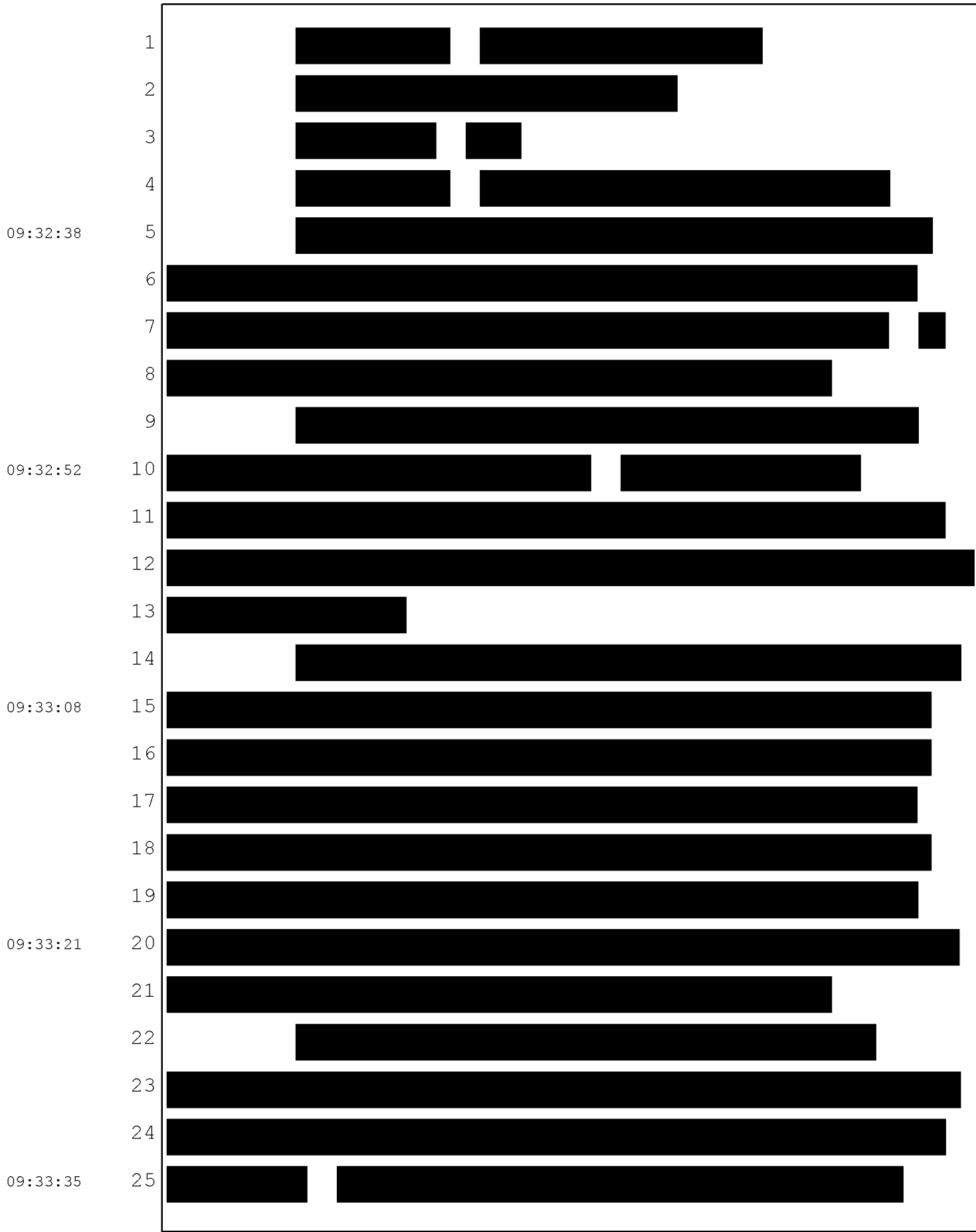
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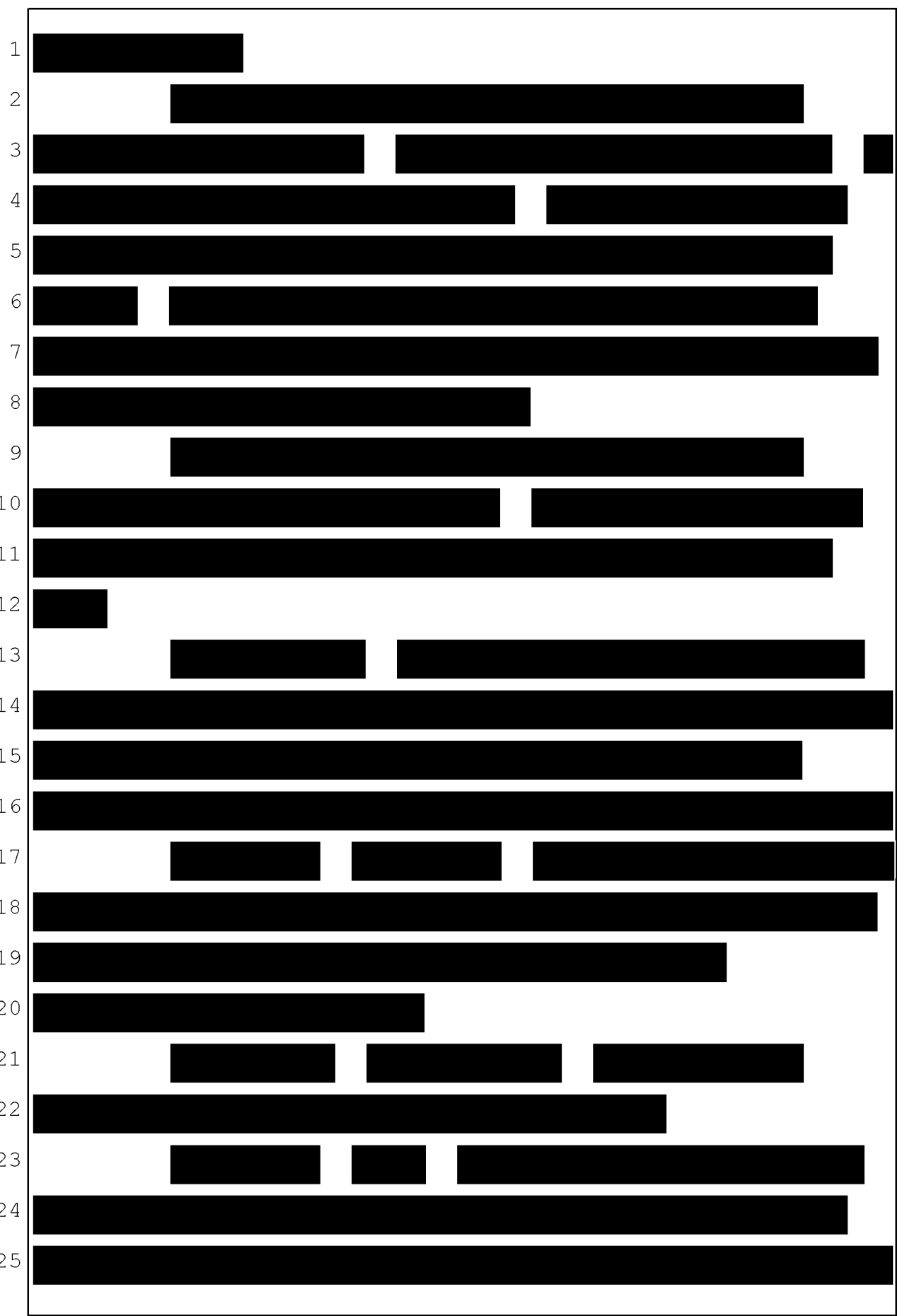
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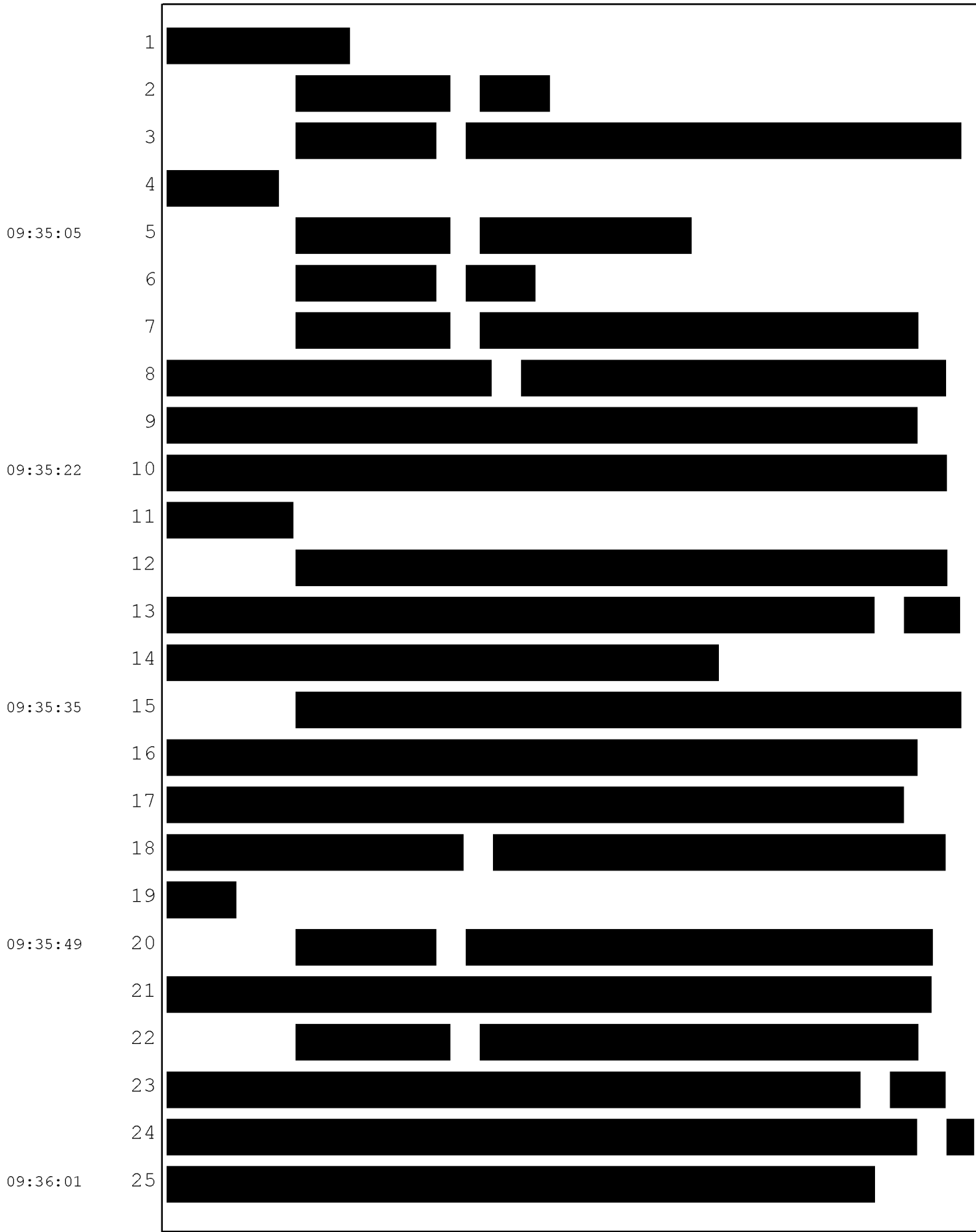
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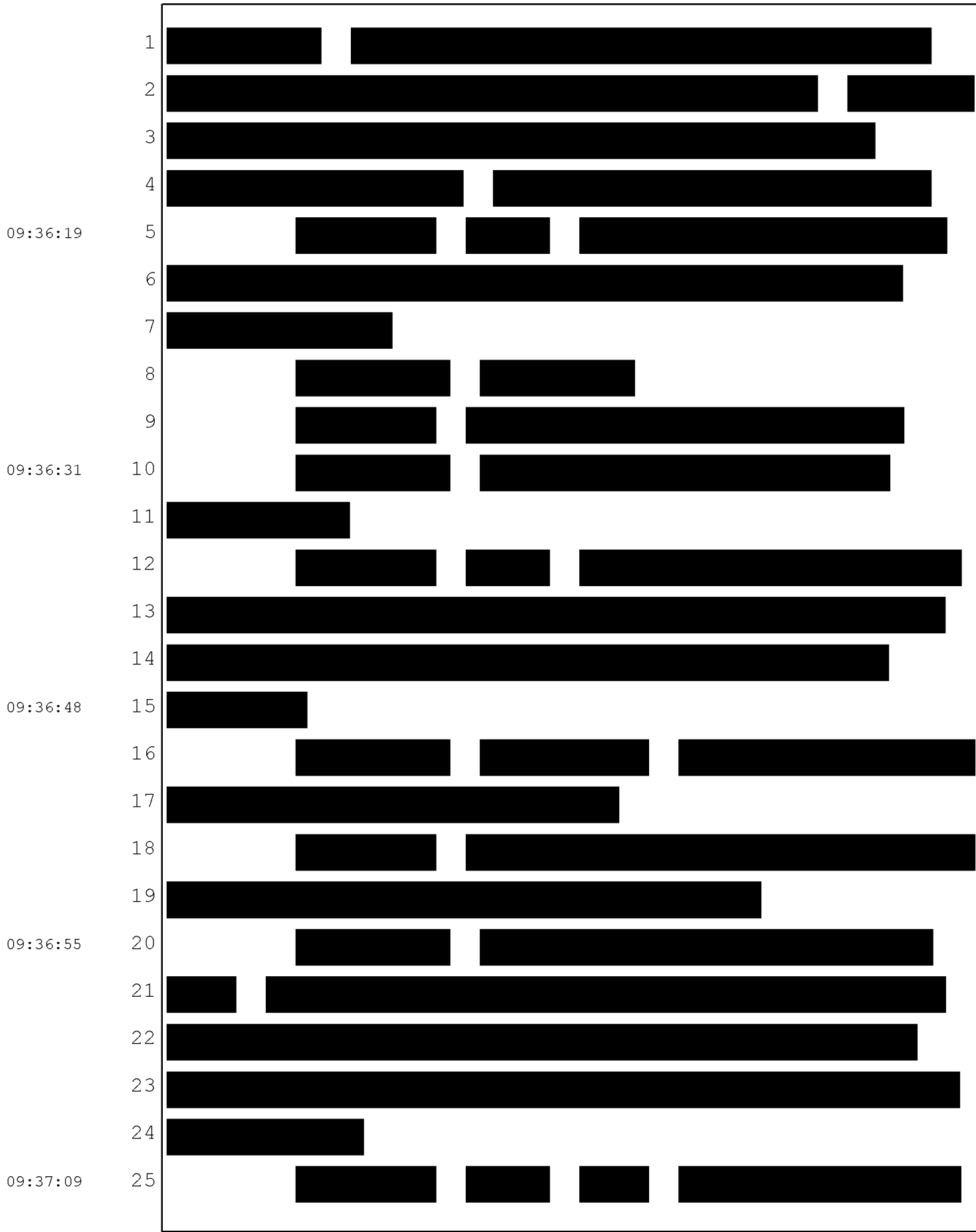
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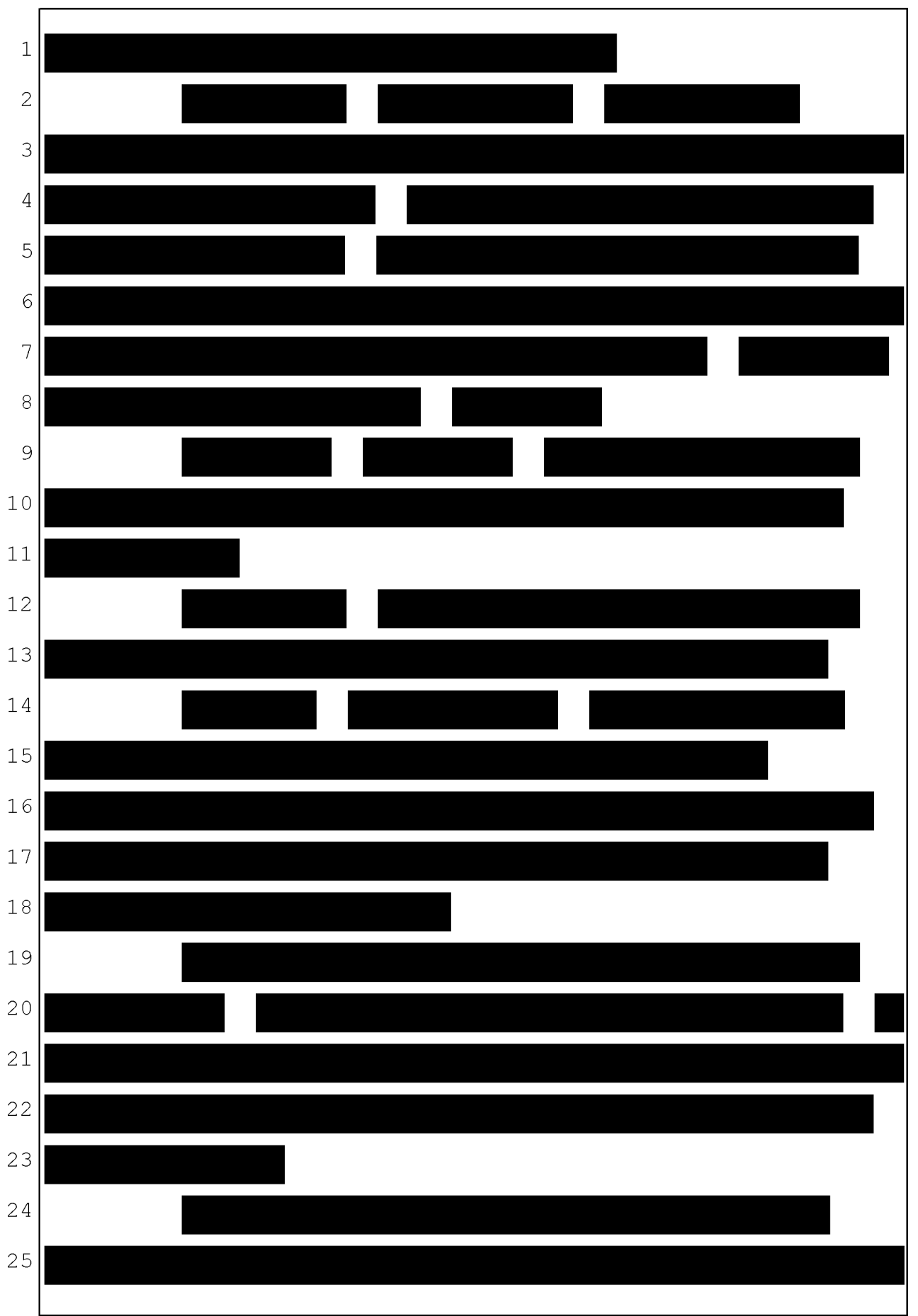
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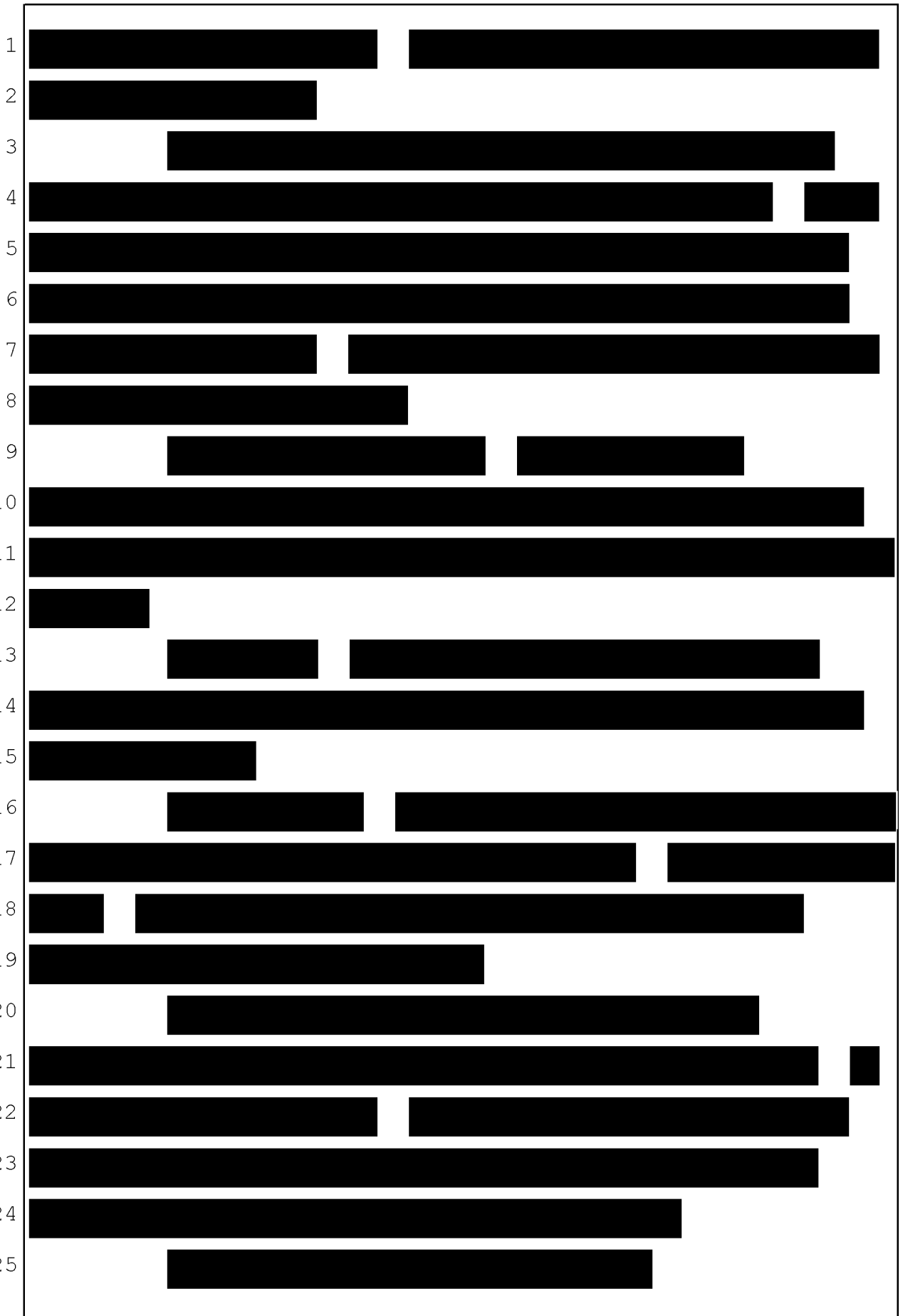
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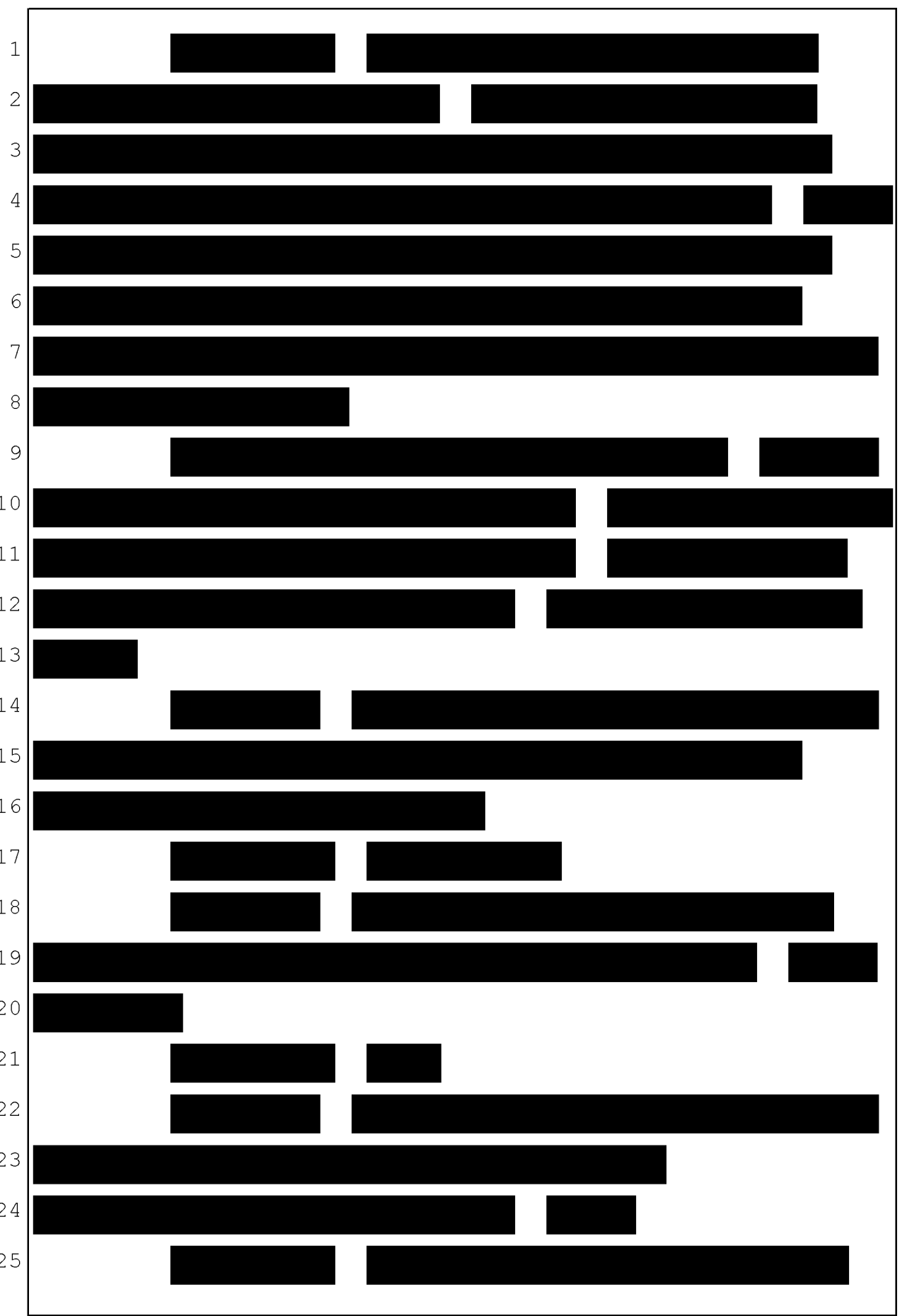
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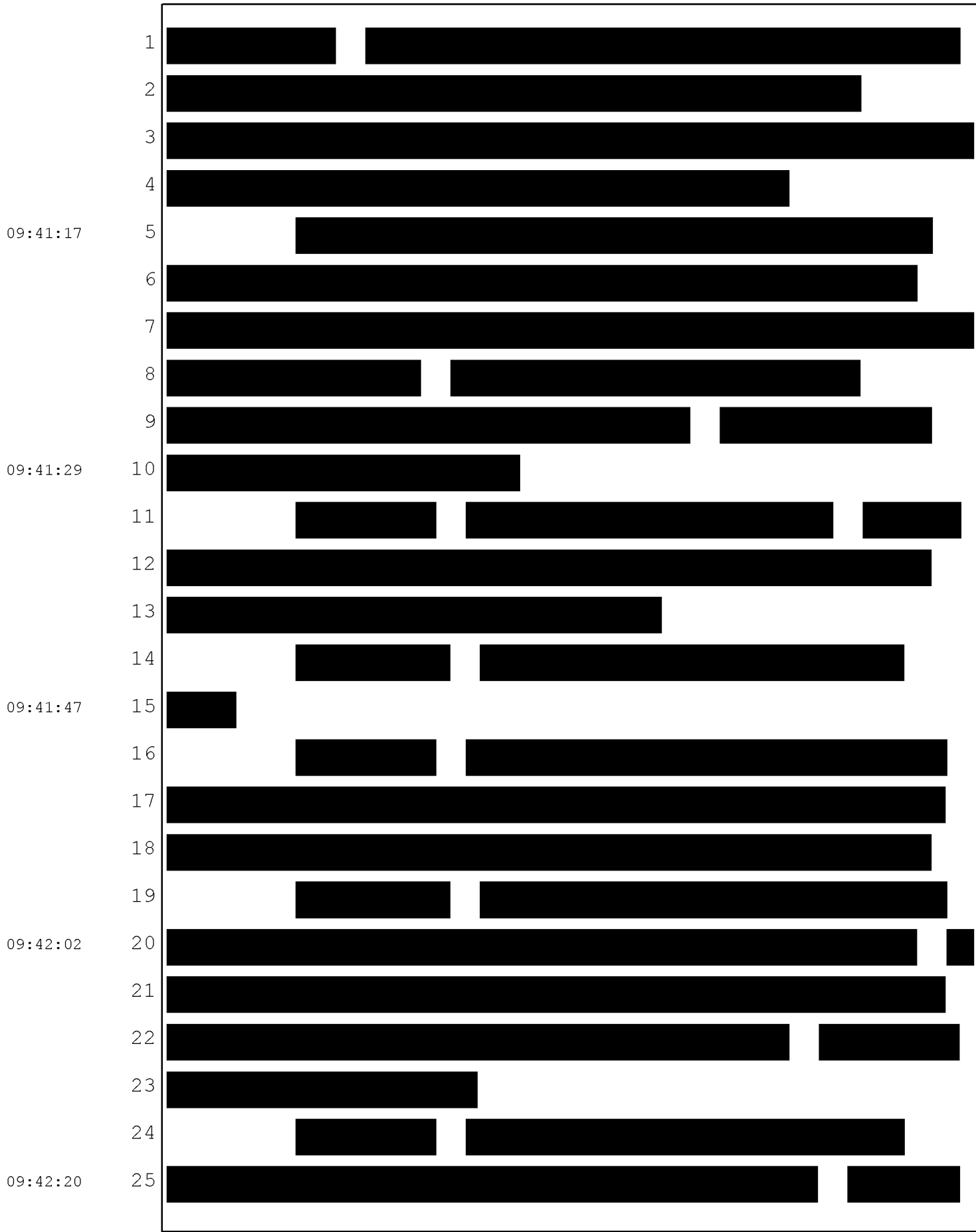
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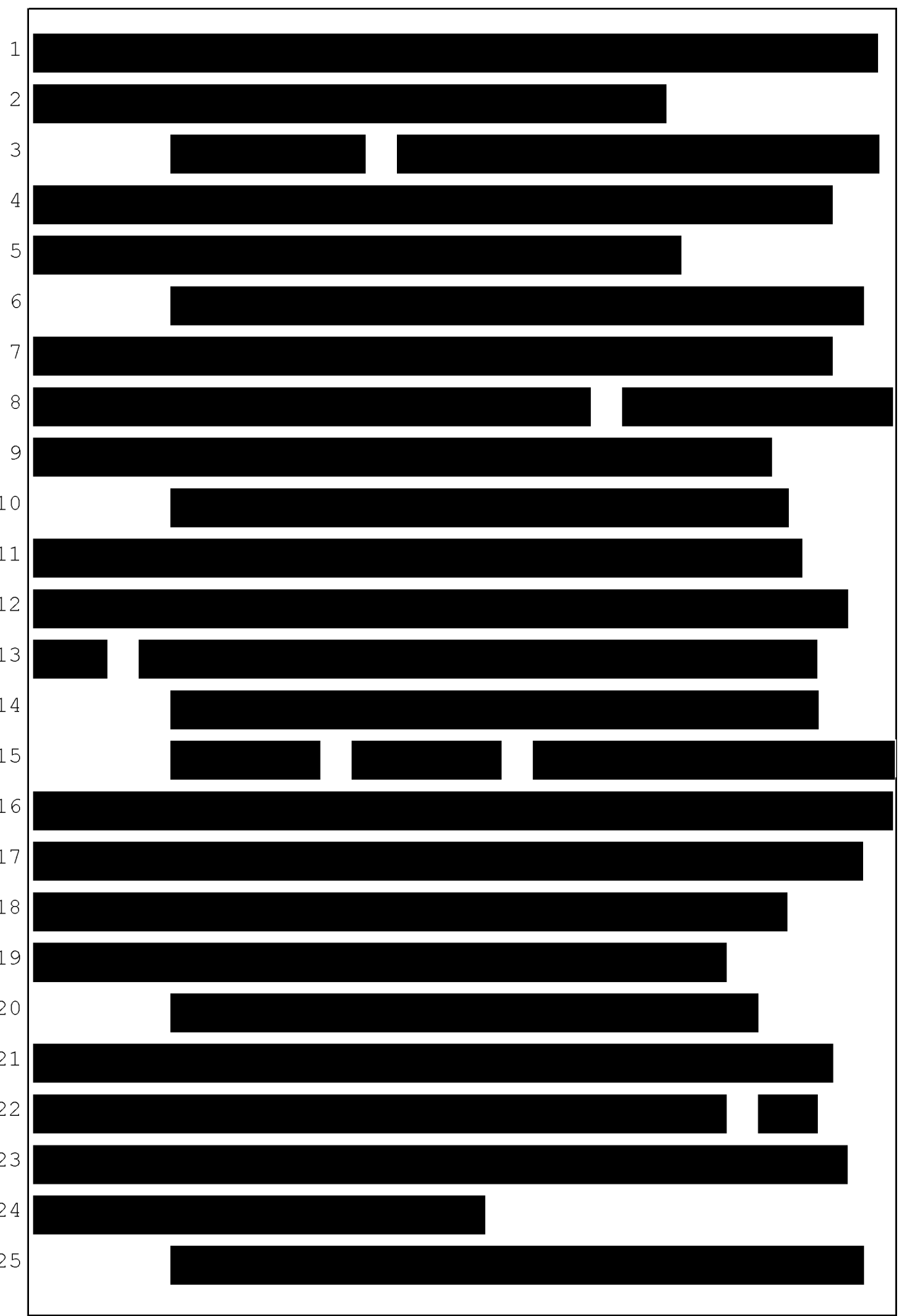
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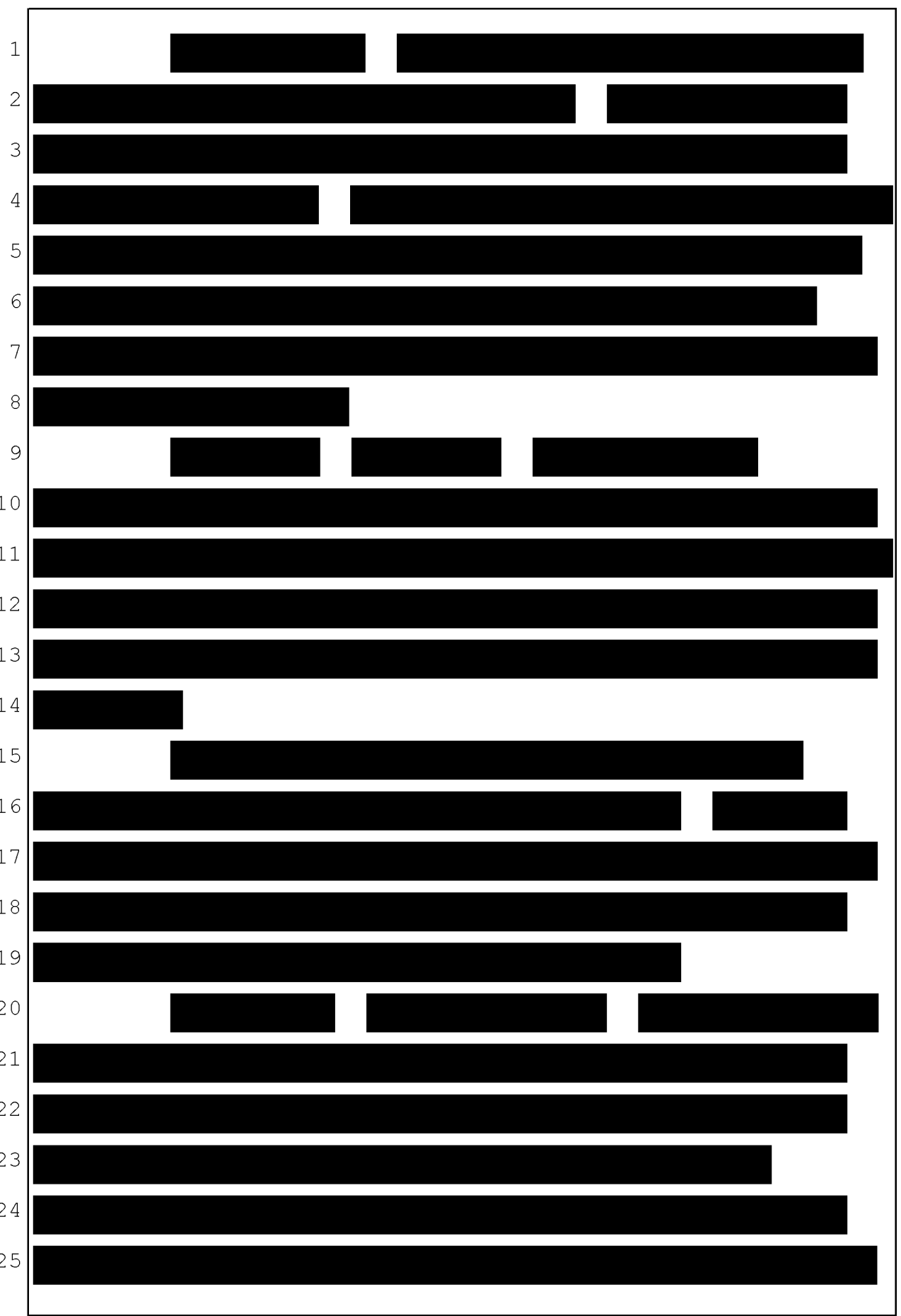
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[REDACTED]

(Jury enters courtroom.)

THE COURT: Good morning, Ladies and Gentlemen.

09:47:32

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.

09:47:53

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:

09:48:35

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:

1 Q. Good morning.

2 A. Good morning.

3 Q. How are you doing?

4 A. Good.

09:48:49

5 Q. Could you please introduce yourself to the jury.

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

09:49:11

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

09:49:27

13 A. Medical oncology is one of the specialties in  
14 medicine that takes care of cancer patients. It's the  
15 one that's primarily involved with giving chemotherapy.

09:49:50

16 I've been treating cancer patients, well,  
17 actually going back to 1980, but as a specialty, and I've  
18 been Board-certified. I trained in it and I still see  
19 patients, which takes up now about I would say 25 to  
20 30 percent of my time, seeing patients.

09:50:08

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

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

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

09:50:25

5 A. Yesterday. I saw about 40 patients yesterday.  
6 They were are -- half of them are -- have potentially  
7 fatal disease and are -- have advanced colon cancer and  
8 are receiving chemotherapy or otherwise being treated for  
9 that.

09:50:40

10 The other half are patients who have had surgery  
11 for locally -- a localized cancer and are receiving or  
12 have received chemotherapy or otherwise being followed  
13 for the disease. And we follow them hoping that it won't  
14 recur, but if it reoccurs to be ready to treat them  
15 further and to make sure that they do -- that they do  
16 okay.

09:51:01

17 Q. Where did you get your medical degree?

18 A. I was -- I went to medical school at Columbia.  
19 I've actually been at Columbia since 1968. I was an  
20 undergraduate at Columbia. I went to Columbia College.  
21 I would say when I got into Columbia, our next-door  
22 neighborhood told my mother, "I'm sorry he couldn't get  
23 into an American school."

09:51:17

24 But subsequently I went to Columbia College, and  
25 from there I went to Columbia Medical School. I was in

09:51:37

1 an M.D. Ph.D. program, which at the time actually -- I go  
2 back so long -- M.D. Ph.D. programs now are fairly  
3 widespread, but back then it was a fairly new thing.  
4 Because I was interested in research, you did research  
09:51:54 5 and clinical training simultaneously.

6           So I did -- I spent two years in a laboratory  
7 doing molecular genetics and chemical carcinogenesis,  
8 studying the chemical carcinogens while I was in medical  
9 school. This again, we're now talking back in the  
09:52:14 10 mid-'70s. This is between 1972 and 1977, when I did my  
11 training for medicine and my Ph.D.

12           I graduated in 1977 with a Ph.D. and an M.D.  
13 The Ph.D. was focused on the growth rates of cancer cells  
14 and also on looking at tumor initiators and promoters and  
09:52:47 15 all of that.

16           From my perspective, the main thing I learned  
17 was that I was a klutz in the lab. Everything I touched  
18 in cell cultures got contaminated, and I learned that I  
19 was not cut out for the research laboratory. That was  
09:53:02 20 not for me.

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

24           So when I graduated medical school, I went on to  
09:53:15 25 do my clinical training. I went on to do a residency in



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

09:53:38 4 At the same time, I -- I had gotten, because of  
5 my laboratory training and my other interests, I was  
6 fascinated by cancer and wanted to do further training  
7 both clinically and otherwise in cancer. So I decided I  
8 would train in -- and do clinical work in oncology.

09:53:55 9 So when it came time to go on from internal  
10 medicine, I decided to specialize in cancer and medical  
11 oncology.

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

13 So I ended up going for clinical training to  
14 Memorial Sloan-Kettering Cancer Center in New York, which  
09:54:14 15 is a cancer center. I trained there in clinical  
16 oncology, learned how to do chemotherapy and learned  
17 about cancer from a clinical perspective.

18 But at the time, from a research point of view,  
19 again, I didn't want to go -- everyone was pushing to go  
09:54:29 20 into a lab, but as I say, that wasn't to me. And at the  
21 time there was a relatively new area in what's called  
22 epidemiology, which I guess we'll talk more about  
23 shortly, but which didn't force me to go into the lab,  
24 where I could do mostly thinking and desk work. So I  
09:54:51 25 wanted to pursue epidemiology and Public Health, which --

1 which is what I pursued.

2           So I left Sloan-Kettering to go -- they did not  
3 have a strong program in that so I went back to  
4 Columbia -- actually, they recruited me for a new program  
09:55:08 5 they had to train clinicians in Public Health and  
6 epidemiology.

7           So I spent two years at Columbia. I went back  
8 to Columbia, where I did a Master's degree in  
9 epidemiology, while at the same time finishing my  
09:55:22 10 clinical training in oncology. Oncology, to do a  
11 sub-specialty in clinical, was three years.

12           So I finished my clinical training in medical  
13 oncology, was Board-certified in medical oncology, while  
14 at the same time doing a Master's degree in epidemiology  
09:55:38 15 learning how you do epidemiology statistics and study  
16 design and things of that sort and learning how to do it.

17           At the time at Columbia -- again, now we're in  
18 the early '80s, '81 to '83 -- there was no person doing  
19 cancer epidemiology. I was basically the only one doing  
09:55:57 20 cancer. Everyone was studying other things from a  
21 research point of view. But I did learn the methods,  
22 which is mainly what you have to learn in epidemiology,  
23 is epidemiology.

24           And so in essence, I became the cancer  
09:56:13 25 epidemiologist at Columbia almost as the only one doing

1 it. And actually, in my second year as a student, there  
2 was a course in cancer epidemiology which I wanted to  
3 take. There was no one to teach it so I taught it. I  
4 enrolled in it, gave myself an A. And I got three  
09:56:35 5 credits for it. And I've actually taught it ever since.  
6 So since 1982, I've taught the course in cancer  
7 epidemiology at Columbia.

8 And --

9 Q. Doctor, let me throw in some questions before I  
09:56:49 10 get an objection.

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

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

09:57:01 15 A. They call it pathobiology -- so because I don't  
16 actually do pathology in the sense of looking at slides  
17 and, you know, deciding what kind of tumor or something  
18 like that, but it's the study of abnormal human biology.

19 Q. And then you also have a Master's in  
09:57:19 20 epidemiology?

21 A. Correct.

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

23 A. Everything's from Columbia.

24 Q. So you've basically been a professor at Columbia  
09:57:27 25 since?

1           A. So I joined -- so when I finished my Master's  
2 degree in Public Health, which was in 1983, and my  
3 clinical training, which was also in 1983, I finished my  
4 fellowship in medical oncology and was Board-certified, I  
09:57:37 5 got a joint appointment at Columbia in medical oncology  
6 and in epidemiology in the School of Public Health. And  
7 so I've had a joint appointment ever since I started as  
8 an assistant professor in medical oncology and in  
9 epidemiology. And then have gradually over the years  
09:57:57 10 risen through the ranks: Assistant professor, associate  
11 professor, full professor.

12           And I actually had a chair, you know, an endowed  
13 chair, in cancer research, which I've had since -- I  
14 don't actually know. I don't remember what year, but  
09:58:17 15 since about 2005, which is a special honor that's given  
16 for achievements in research and whatever.

17           So -- so I'm basically now my rank is -- or my  
18 title is Myron M. Studner, Professor of Cancer Research  
19 and Professor of Medicine in Epidemiology at Columbia.  
09:58:36 20 And then I have various other titles, but those are the  
21 main ones that I talk about.

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

09:58:51 25           How many peer-reviewed journal articles have you

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

2       A. Well, if you say "the causes of it," I'm not  
3 exactly sure how many papers. I've published over 600  
4 papers. I would say of those papers, if we took just  
09:59:10 5 about causation, probably 3- or 400 have some tie to  
6 causation.

7       But I have other interests in terms of, for  
8 example, my research more recently, say since about 2000  
9 or 2003, has been on quality of care for cancer patients.  
09:59:31 10 So I do a lot of research using epidemiologic methodology  
11 to study what's called health outcomes research, which is  
12 the quality of care patients get, who, if you're supposed  
13 to get chemo for certain type of -- in certain contexts,  
14 does everyone get it. If they don't get it, why don't  
09:59:51 15 they get it? Are they too poor? Are there racial  
16 disparities? Are there gender differences and things  
17 like that.

18       And again, this uses the same types of  
19 epidemiologic methodology as is used in etiologic  
10:00:11 20 research. So it's really an epidemiologic type of  
21 research. That's now one major area of my research  
22 efforts.

23       The other major area of my research -- my  
24 current research efforts is since about 2007 or 2008,  
10:00:32 25 for -- someone -- someone gave -- some agency gave money

1 to Columbia to study HIV and cancer -- or to study HIV  
2 and cancer in Africa, where the -- there's high rates  
3 of -- obviously of high prevalence of HIV in sub Saharan  
4 Africa. And no one else knew anything about it so they  
10:00:54 5 asked me to do it.

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

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

14 Cancer rates are going up in sub Saharan Africa  
10:01:33 15 because we're now giving out antiretroviral therapy to  
16 people with HIV. Before, the life expectancy in South  
17 Africa or in other countries in sub Saharan Africa was  
18 very low because of HIV. So people weren't living past  
19 50 or 55, and so they weren't getting the cancers that we  
10:01:52 20 normally associate with in the West -- breast cancer,  
21 colon, prostate. They didn't live long, and those are  
22 diseases of middle and older ages, but now they're  
23 getting them more so it's becoming a major problem in sub  
24 Saharan Africa. And so we're studying how that interacts  
10:02:07 25 with HIV.

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

4 Q. All right, Doctor. You know, we could go  
10:02:19 5 through your CV for literally two hours, but we have a  
6 two-hour clock here. So I'm just going to put it  
7 together.

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

10:02:30 10 THE COURT: Any objection?

11 MR. LOMBARDI: No objection.

12 THE COURT: Very well.

13 Q. BY MR. WISNER: All right, Doctor, we're looking  
14 here on the screen. This is a copy of your CV. We can  
10:02:36 15 zoom in here. This is actually a bit old. It's as of  
16 April 1st, 2017.

17 Do you see that, Doctor?

18 A. Yes.

19 Q. It goes through all your -- just to give you  
10:02:44 20 some context, Doctor, my résumé is one page. So let's go  
21 through this quickly.

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

10:03:00 25 A. Yes.

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

4 A. Yes.

10:03:06

5 Q. And I know this stops at 2016. I understand you  
6 recently achieved -- received a lifetime achievement  
7 award; is that right?

8 A. Yes, uh-huh.

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

10:03:18

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

14 So two years ago they gave me their  
15 distinguished achievement award, which is given out every  
16 year to I guess someone who has had distinguished  
17 achievement.

10:03:33

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

10:03:48

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

10:04:08

25 A. I would ballpark it at around somewhere in the



1 45 to \$50 million range.

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

10:04:20 5 A. Yes, all related to cancer.

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

9 A. Yes.

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

13 A. Uh-huh.

14 Q. -- since I guess it was created?

10:04:43 15 A. Uh-huh.

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

18 Do you see that?

19 A. Yes.

10:04:50 20 Q. And so these are people you've supervised for  
21 their doctoral as well as Master's theses?

22 A. Yes.

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

10:05:02 25 Do you see that, Doctor?

1 A. Yes.

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

10:05:08 5 A. Post-doctoral training is someone who has gotten  
6 a Ph.D. and in the past -- go back, I don't know, 10 or  
7 15 years, maybe after a Ph.D. you could actually get a  
8 job. Nowadays that's becoming more difficult.

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

10:05:45 15 So they do a post-doc, working with some senior  
16 or someone at some academic institution to do more  
17 research or maybe something different so they get a  
18 little more experience and then go on to do that.

19 And so I've been lucky enough to have a lot of  
10:06:03 20 post-docs train with me, and they've all -- many of them  
21 have gone on to have their own academic -- successful  
22 academic careers.

23 Q. All right. I'm going to sweep through this.  
24 You have you've worked on an editorial board. So I take  
10:06:21 25 it you've reviewed other people's published literature to

1 see if it was appropriate?

2 A. We see manuscript reviewers. So, you know, we  
3 do peer review. That's if you publish a lot of papers,  
4 they're also going to ask you to do peer review for other  
10:06:36 5 people's papers. So I end up doing a lot of peer review  
6 for various journals.

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

9 A. Yes.

10:06:47 10 Q. Oh, look at that.

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

14 Do you see that?

10:07:00 15 A. Yes.

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

18 A. Yes.

19 Q. You have state and local. We have study  
10:07:06 20 sections. We have private foundations, international,  
21 other, and then we start the publications.

22 Do you see that's on page 20?

23 A. Yes.

24 Q. Okay. And then this goes on, I mean, for  
10:07:19 25 hundreds. So let's go through this quickly. All right.

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

3 A. Sometimes a journal will ask you to -- actually  
4 will invite you to write a chapter or a review article on  
10:07:38 5 some subjects. So opposed to you having to write it up  
6 yourself and then submit it and it will be accepted or  
7 whatever.

8 Q. Okay. We have a few of those, and we have book  
9 chapters, quite a few of those. It goes on to page 65  
10:07:55 10 with editorials. It goes on for a few pages. We have  
11 books and letters.

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

10:08:09 15 Doctor, have you looked at herbicides and its  
16 effects on cancer?

17 A. I'm sorry.

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

10:08:17 20 A. So I don't myself study it as a researcher, but  
21 this book reflects a committee that was established in  
22 the early '90s by the Institute of Medicine. It was  
23 asked by the Veterans Administration at the time to  
24 review for the purpose of deciding whether a Vietnamese  
10:08:46 25 veteran -- not Vietnamese, but --

1 Q. Vietnam.

2 A. Yes. That veterans of the Vietnam War, who were  
3 exposed to Agent Orange, there was literature that  
4 suggested that because of exposure to Agent Orange, they  
10:09:01 5 were at an increased risk of cancers and other diseases,  
6 other problems.

7 So the VA was interested or wanted to know  
8 whether they were obligated to compensate them for these  
9 injuries stemming from Agent Orange exposure from the  
10:09:16 10 herbicide spraying in -- that took place in Vietnam. So  
11 they established this committee of experts, not  
12 dissimilar to the -- it was actually very similar to the  
13 way the IARC committees are established.

14 So I was on that committee. I was the chair of  
10:09:35 15 the cancer subcommittee within this -- within this  
16 committee.

17 So the subsequent report was actually published  
18 as this book in 1994 and found that there were certain  
19 cancers that stemmed from being exposed to Agent Orange  
10:09:55 20 and that -- and I guess the recommendation was that the  
21 VA should compensate Vietnam veterans who were exposed to  
22 Agent Orange and developed these specific cancers  
23 subsequently.

24 Q. All right. Then we have letters, invited  
10:10:10 25 presentations. That goes on for a few pages. And that's

1 the end, page 75 of your CV?

2 A. Uh-huh, yes.

3 MR. WISNER: At this time, your Honor, I'd like  
4 to have Dr. Neugut recognized as an expert in the area of  
10:10:24 5 medical oncology and cancer epidemiology.

6 MR. LOMBARDI: No objection.

7 THE COURT: Any *voir dire*?

8 MR. LOMBARDI: No objection, your Honor.

9 THE COURT: Very well. Then I will accept

10:10:32 10 Dr. Neugut as an expert in the areas of medical oncology.  
11 And Counsel, did you ask for cancer epidemiology?

12 MR. WISNER: Yes, your Honor.

13 THE COURT: And cancer epidemiology.

14 All right. Thank you. You may proceed.

10:10:50 15 Q. BY MR. WISNER: All right, Doctor. What were  
16 you asked to do in this case?

17 A. I'm sorry.

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

19 A. I was asked to review the literature and to  
10:11:03 20 opine on the association between Roundup or glyphosate  
21 and its association with non-Hodgkin's lymphoma.

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

10:11:21 25 A. So when I was -- so whenever I'm asked to be an

1 expert or to comment on a case, I do my own literature  
2 review initially before I will accept a case, because  
3 obviously I don't want to participate in a situation  
4 where I'm not comfortable or where I don't know that it's  
10:11:49 5 true or whatever. So I usually do sort of my own  
6 literature review off the record sort of uncompensated.

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

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

12           A. I would say that within the scientific and  
13 academic cancer community, IARC is recognized as the main  
14 arbiter of -- the prime arbiter of what constitutes a  
10:12:40 15 carcinogen or a cancer-causing agent. I would have  
16 trouble even naming a second -- I would have trouble  
17 naming a second choice.

18           Now, you do have sometimes the problem that IARC  
19 hasn't reviewed -- you often have the problem that IARC  
10:12:57 20 hasn't reviewed a particular agent, and then obviously in  
21 that context, you don't have an IARC Monograph or an IARC  
22 publication to use as a -- to help you.

23           But when there's an IARC Monograph, then I would  
24 say almost uniformly that's what everyone -- and I can't  
10:13:22 25 speak for everyone; I can speak for myself and I can

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

4 Q. You don't go to the EPA?

10:13:36

5 A. No.

6 Q. Why not?

7 A. It never crossed my mind.

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

10:13:48

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

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

10:14:01

15 Q. Okay. Well, hold on a second.

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

18 MR. LOMBARDI: No objection, your Honor.

19 THE COURT: Very well. You may proceed.

10:14:13

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

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

24 Q. And walk us through what this document is and

10:14:25

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



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

3 A. Can you?

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

10:14:40 5 A. Thank you. So before I even address this, let  
6 me tell you what epidemiology is more broadly before I  
7 address the -- because this is really more intended to  
8 tell you how you do epidemiology than what epidemiology  
9 is.

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

16 When I started out everyone thought --  
17 epidemiology wasn't as well known. Everyone thought it  
18 came from the word epidermis, and they thought I was  
19 studying skin. But in truth, epidemiology is what I just  
10:15:33 20 said.

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

10:15:47 25 But the purpose of epidemiology is for Public

1 Health purposes. It's not so much intended for medicine  
2 for treatment, but for Public Health in order to reduce  
3 its incidence in the population, in order to prevent the  
4 disease from occurring. If we figure out why you get the  
10:16:09 5 disease, then we can take steps to prevent the disease.

6 So the purpose of epidemiology, the underlying  
7 purpose, is to figure why people get heart disease, colon  
8 cancer, whatever disease, HIV, et cetera, to figure out  
9 why they get it. And to do that is not -- often not very  
10:16:28 10 easy to achieve and through various methodologies.

11 So going to this board that sets us up for this,  
12 so in most of science, in fact -- this is a pretty good  
13 layout for science -- we're mostly studying in most of  
14 science is the association between an exposure and an  
10:16:54 15 outcome. Does tobacco smoking cause lung cancer? Does  
16 taking Lipitor reduce your incidence of heart disease?  
17 Does putting salt in a solution increase the release of  
18 heat from a solution?

19 This is the underlying -- you could say this is  
10:17:14 20 the underlying phenomenon of science. This is for all of  
21 science. But in the context of epidemiology, we're --  
22 what we're talking about today, the exposure and the  
23 outcome is usually some exposure, whatever it might be --  
24 obviously, in our case we're talking about Roundup or  
10:17:34 25 glyphosate -- and an outcome, which in our case today of

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

3           And as in all of science -- as in all of  
4 science -- and epidemiology is no different than all of  
10:17:53 5 science -- we start off with what's called the null  
6 hypothesis. And to put it in CSI terminology, or Law and  
7 Order, which is my favorite show, we start off innocent  
8 until proven guilty. We assume that they're random. In  
9 other words, the underlying assumption is that the  
10:18:14 10 exposure and the outcome have nothing to do with each  
11 other.

12           And it's our job, our duty or whatever you want  
13 to call it, our underlying goal is to assess, to do  
14 studies to determine whether the exposure and the outcome  
10:18:29 15 have something to do with each other, they're nonrandom  
16 document. But the underlining is we start off  
17 everybody's innocent until proven guilty.

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

10:18:43 20           So the studies that we do in epidemiology are to  
21 find nonrandomness or to see if there is nonrandomness,  
22 if the two really are linked together.

23           *A priori*, we say tobacco and lung cancer have  
24 nothing to do with each other, but when we do studies and  
10:19:01 25 we find that they are linked to each other, then we say

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

10:19:09

5           So what an epidemiologic study does is, an  
6 epidemiologic study does not tell you that an exposure  
7 causes an outcome. An epidemiologic study tells you that  
8 the exposure and the outcome are statistically associated  
9 and nonrandom, that they occur together more commonly

10:19:26

10 than -- than would generally be the case in a general --  
11 randomness would have asserted.

12           It doesn't mean they're causal. It just means  
13 that they're statistically associated more. They occur  
14 together more commonly than -- smokers and lung cancer  
15 occur together more frequently than we would expect. It  
16 doesn't mean tobacco -- maybe having lung cancer makes  
17 you want to smoke because it makes your lungs feel  
18 better, for all you know. Could be. But just the fact  
19 is it's nonrandom.

10:19:54

20           And how do we do this in the epidemiologic  
21 study? Because only two types. Easy peasy. There's  
22 only two types of epidemiologic studies that exist.  
23 There's only two types. Easy. You either go this way or  
24 you go this way. No other study.

10:20:13

25           So if you start from the outcome, you say I'll

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

4 Q. 100 people with lung cancer.

10:20:34

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

10:20:51

13 So that's one type of epidemiologic study.  
14 That's called the case-control study. That started with  
15 cases, lung cancer cases and control, people who don't  
16 have lung cancer. And that's called a case-control  
17 study. I started from the outcome, from the lung cancer,  
18 and I went back to the exposure. I asked how many smoke  
19 and how many don't smoke.

10:21:12

10:21:28

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

23 THE COURT: Excuse me, Doctor.

24 Mr. Wisner, can I please remind you to proceed  
25 by way of question and answer.

10:21:41

1 MR. WISNER: Oh, sorry.

2 THE WITNESS: Sorry. That's his job.

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

10:21:53

5 MR. WISNER: I think we have to do one more  
6 direction and he'll sit down.

7 THE COURT: Okay, great.

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

10:21:58

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

12 A. Okay.

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

14 A. Right. So the other study is going forward,

10:22:07

15 which is to start from people who smoke and people who  
16 don't smoke and go forward to asking -- starting with  
17 people who smoke and people when don't smoke, and ask how  
18 many of each of them, if I take a large number of each,  
19 and ask how many of them get lung cancer in each group.

10:22:24

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

24 And again, I'll let the attorneys ask about

10:22:40

25 whether -- how that relates to our current case. And

1 that's called a cohort study.

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

10:22:58

5           That's all of -- but again, these -- I'll sit  
6 down, your Honor.

10:23:16

7           Either one of these studies, the case control or  
8 the cohort study, on its own only tells us that there's a  
9 statistical association between the two; that it's not,  
10 as I said, nonrandom between the two. It doesn't tell us  
11 if there is actual -- or that we might assume there is if  
12 it's very dramatic, someone might want to infer that.  
13 But on its own, it only tells us -- it only tells us  
14 there's a statistical association.

10:23:35

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

10:23:55

19           A. So for that I have to riff a little bit on the  
20 question of how you establish causation for a moment,  
21 which is -- it is harder -- the more rare the outcome is,  
22 the more rare the exposure is makes it more difficult to  
23 establish an association. And certainly if the two of  
24 them are uncommon, it becomes much more difficult to  
25 establish these associations just statistically and

10:24:24

1 methodologically.

2           And if I can illustrate that, I'll say, for  
3 example -- well, let me back up and talk a little bit  
4 about causation because there you can see it a little  
10:24:40 5 better, if I may.

6           Q. Please.

7           A. So the whole question of how to establish  
8 causation is an issue. So how to establish causation  
9 goes back to Hippocrates or the Greeks, who talk about  
10:24:58 10 it, and all the medieval philosophers talk about how to  
11 establish causation. Establishing causation is a very  
12 difficult phenomenon over and above statistical  
13 association. And the question is how do we establish  
14 causation in general in our lives.

10:25:16 15           The answer is in a weird way. It's by --  
16 generally speaking, it's by what I would call inductive  
17 reasoning. A child goes around, a toddler, and flips a  
18 light switch, let's say, in the kitchen, and the light  
19 goes on. The child doesn't necessarily put flipping the  
10:25:35 20 light switch with the light going on. Doesn't  
21 necessarily make the connection. But if he does it two,  
22 three, four, five times, after awhile, and every time the  
23 kid flips the light switch, the light goes on. After  
24 four, five, six times and the kid's smart enough, if it's  
10:25:53 25 your kid, the kid will understand finally that flipping



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

4           Now let's say we want to play with the kid's  
10:26:10 5 head, and we'll make it that randomly the light will only  
6 go on 50 percent of the time when the light switch goes  
7 on and it will be random.

8           So now the kid flips the switch. Sometimes the  
9 light goes on, sometimes the light doesn't go on. And  
10:26:18 10 the kid's going to be, like, uh-huh.

11           And so it's going to take now instead of maybe  
12 four or five or six times, maybe it will take eight or  
13 ten or twelve times, but after ten, twelve times, if it's  
14 only 50 percent, the kid's finally going to understand  
10:26:34 15 still that there's still a causal connection between  
16 flipping the light switch and the light going on,  
17 although there's something screwy about the light switch  
18 and the light going on.

19           Let's say, I made it one in ten times, one in  
10:26:47 20 ten times when you flip the light switch. We can do  
21 this, you know, and drive someone nuts.

22           But let's say you do it one in ten times, the  
23 light's randomly going to go on. It's a kid, it's a  
24 five-year old is going to figure out that the light  
10:27:02 25 switch is connected to the light going on. It would

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

3           So as you make it more and more unlikely of the  
4 probability of the light going on, more and more  
10:27:18 5 uncommon, it's going to be harder and harder for a child  
6 to establish a causal connection between the phenomenon  
7 and the outcome -- between the exposure, the flipping the  
8 switch, and the outcome.

9           Q. Now, Doctor, you're talking one out of ten.

10:27:33 10 We're talking about cancer like non-Hodgkin's lymphoma --

11           A. Right.

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

13           A. So most of cancer epidemiology, if you actually  
14 look at the literature and you see what I do every day or  
10:27:46 15 most of my colleagues do every day, is focused on the  
16 four most common cancers, which are breast, prostate,  
17 colon, and lung. They occur in an incidence rate of one  
18 in a thousand per year. And so most studies in colon  
19 cancer -- in cancer will focus on those four cancers,  
10:28:03 20 which are very common.

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

24           If we talk about non-Hodgkin's lymphoma, the  
10:28:16 25 topic of the day, then we're talking about two in 10,000

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

3           And if I put on top of that that we're talking  
4 now about -- at least in the context of Roundup -- a  
10:28:37 5 relative risk of about 1.4 to 1.5, so we're talking about  
6 a 50-percent increase of the incidence of the disease.  
7 So we're talking about going from two in 10,000 to three  
8 in 10,000.

9           So that is a very difficult phenomenon to  
10:29:00 10 establish using epidemiologic methodology. And that is  
11 why -- that's in part why we're here today, but that's a  
12 very difficult thing to establish through epidemiologic  
13 studies, but not impossible. And it's difficult and  
14 makes it -- it means that any -- a small error, any small  
10:29:19 15 phenomena are going to have a profound effect on what you  
16 observe in the epidemiologic studies. Because we're  
17 talking about a very relevant natively small delta --  
18 relatively small difference to be observed.

19           And to try to establish with evidence, as I said  
10:29:40 20 before, innocent -- the entire epidemiologic methodology  
21 is conservative. All of science is conservative. We  
22 don't want to find a positive finding when there isn't  
23 one. We don't want to implicate an innocent person, an  
24 innocent exposure, as being guilty when it's not.

10:30:01 25           So the entire statistical and epidemiologic

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

4           And so when you don't see a positive  
10:30:31 5 association, you cannot be certain if you don't see the  
6 association, either because it's truly innocent, truly  
7 negative or null, or because all the biases have made it  
8 null or biased the results in that direction.

9           When you see a positive finding, then you can  
10:30:49 10 probably have a lot more confidence because, again, we're  
11 more -- we've set the system up to make the positive  
12 finding the more robust phenomenon.

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

10:31:11 15           MR. WISNER: Permission to approach the witness  
16 with the binder.

17           THE COURT: Yes.

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

10:31:17 20           THE WITNESS: Okay.

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

24           A. Okay.

10:31:41 25           Q. And this is a journal article. First author is

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

3 A. Yes.

4 MR. WISNER: Your Honor, permission to publish.

10:31:51

5 THE COURT: Any objection?

6 MR. LOMBARDI: Just checking something, your  
7 Honor.

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

10:32:10

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

11 MR. WISNER: Here (indicating).

12 (Sidebar.)

13 [REDACTED] [REDACTED]  
14 [REDACTED] [REDACTED]  
15 [REDACTED]  
16 [REDACTED]  
17 [REDACTED]  
18 [REDACTED]

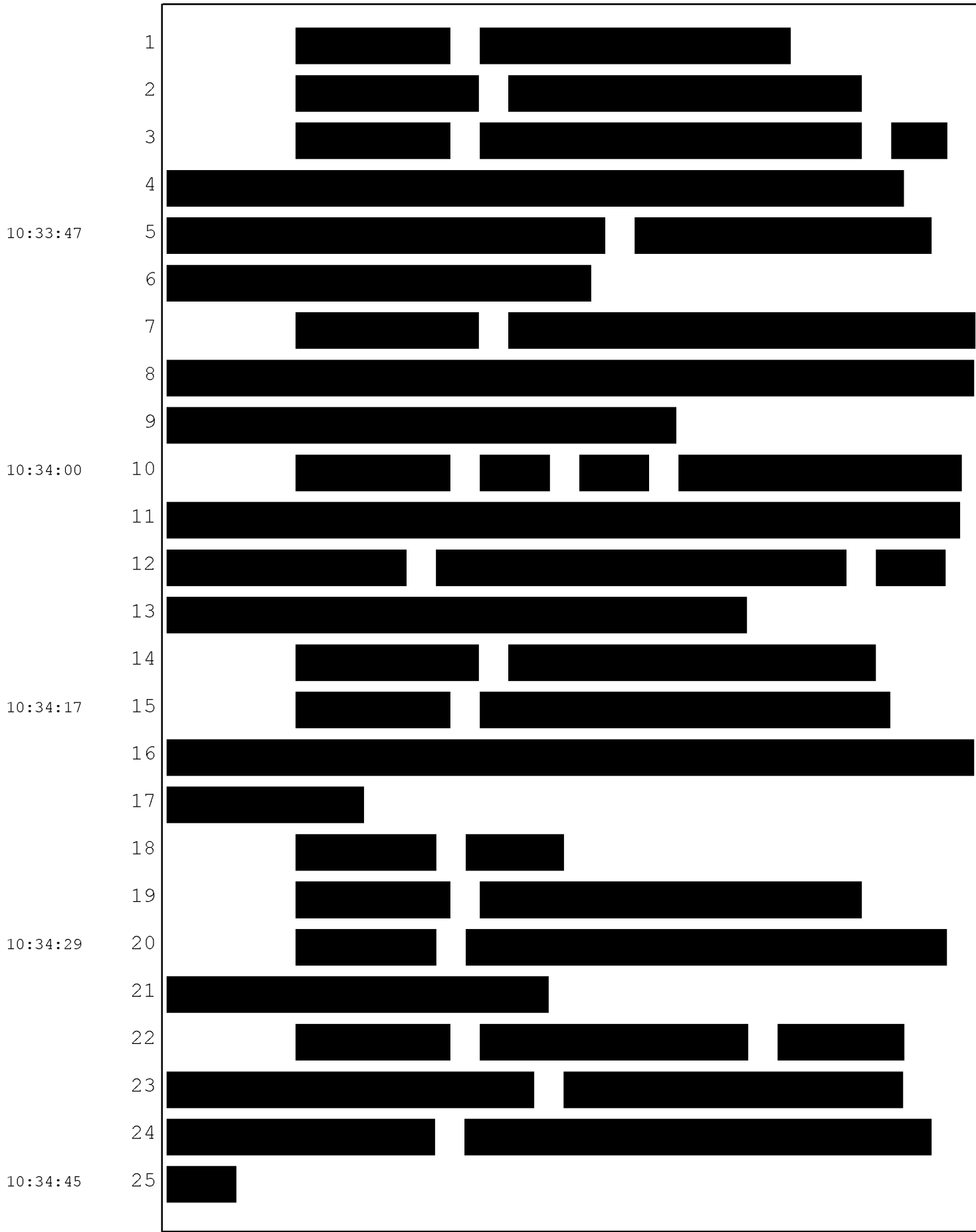
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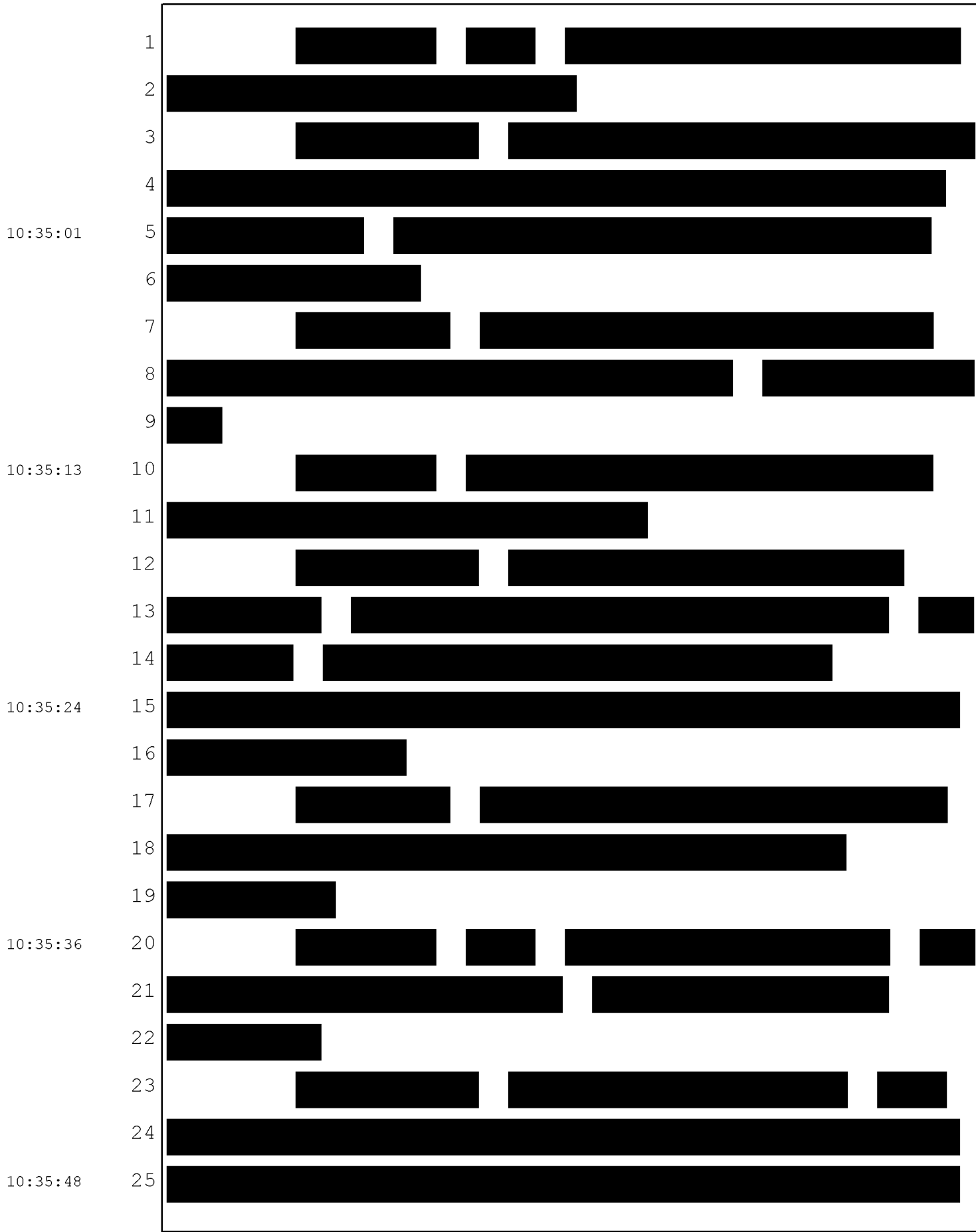
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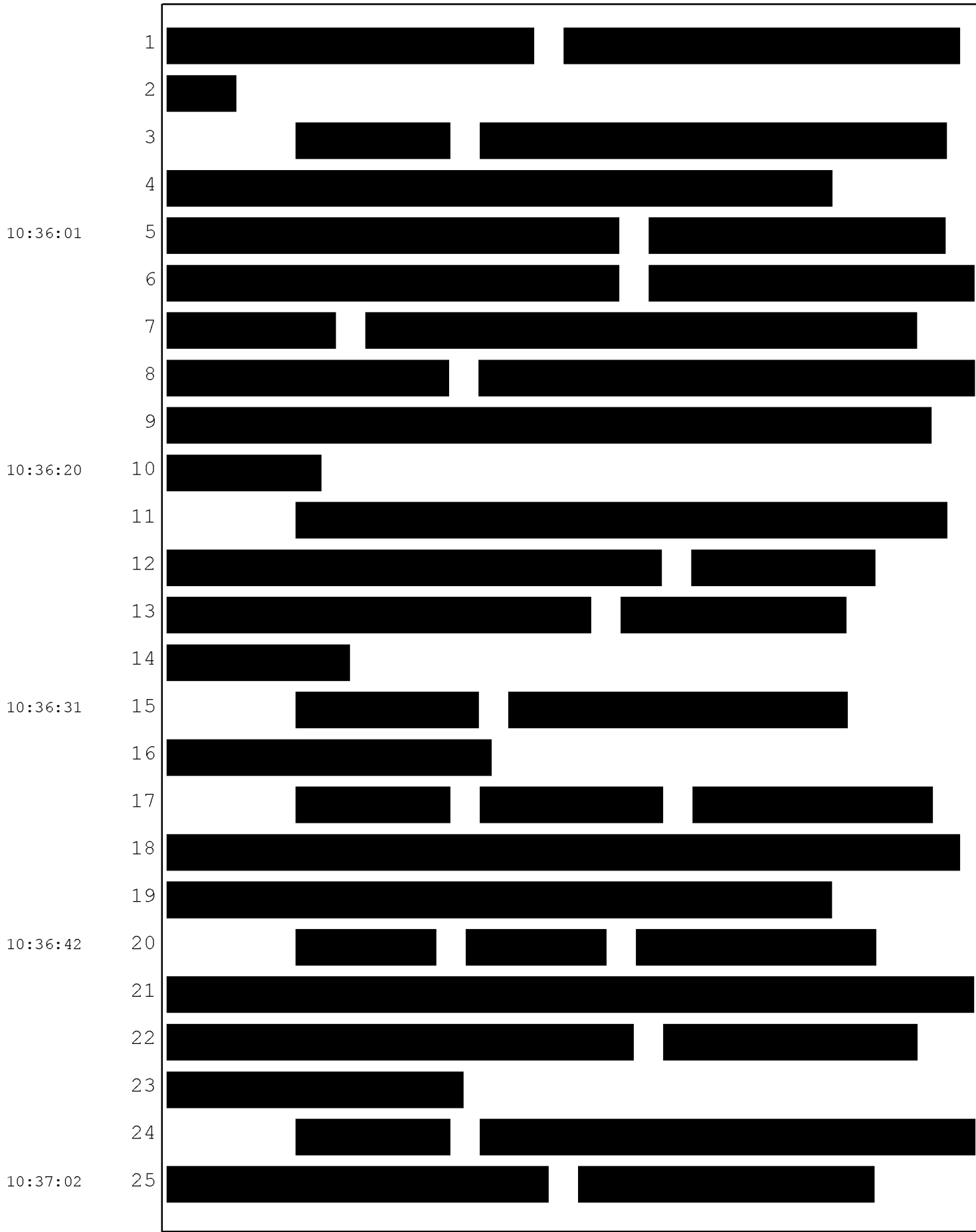
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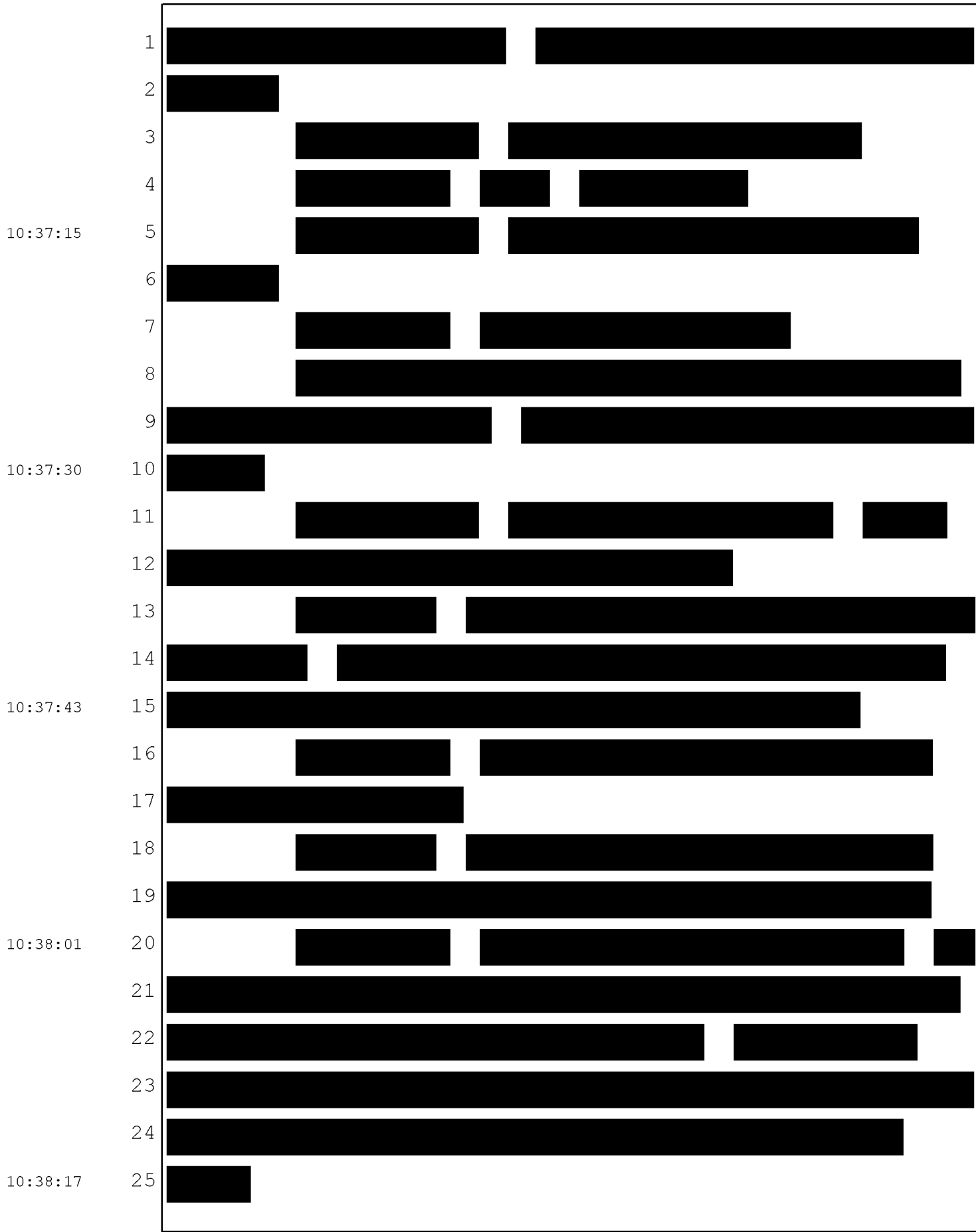
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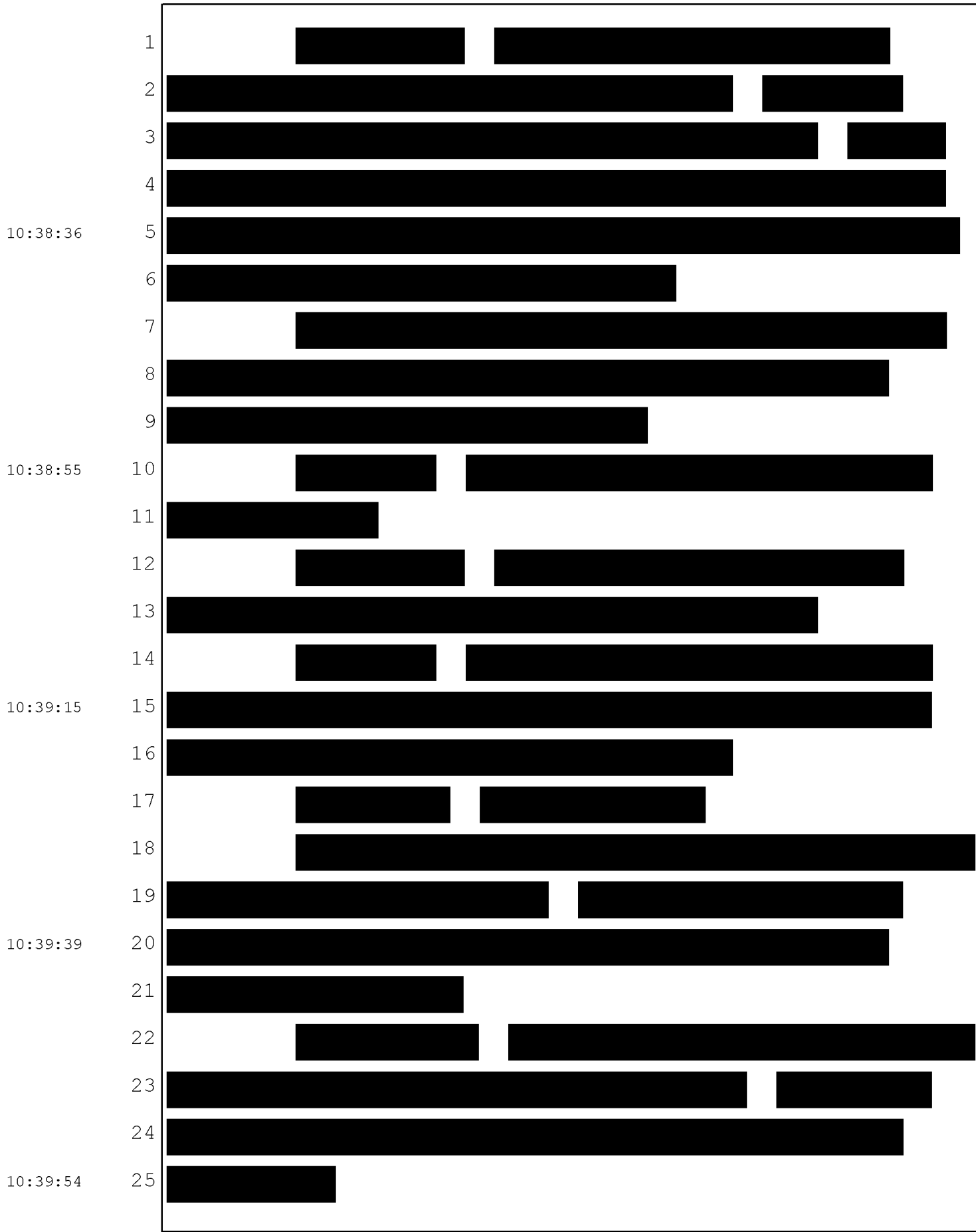


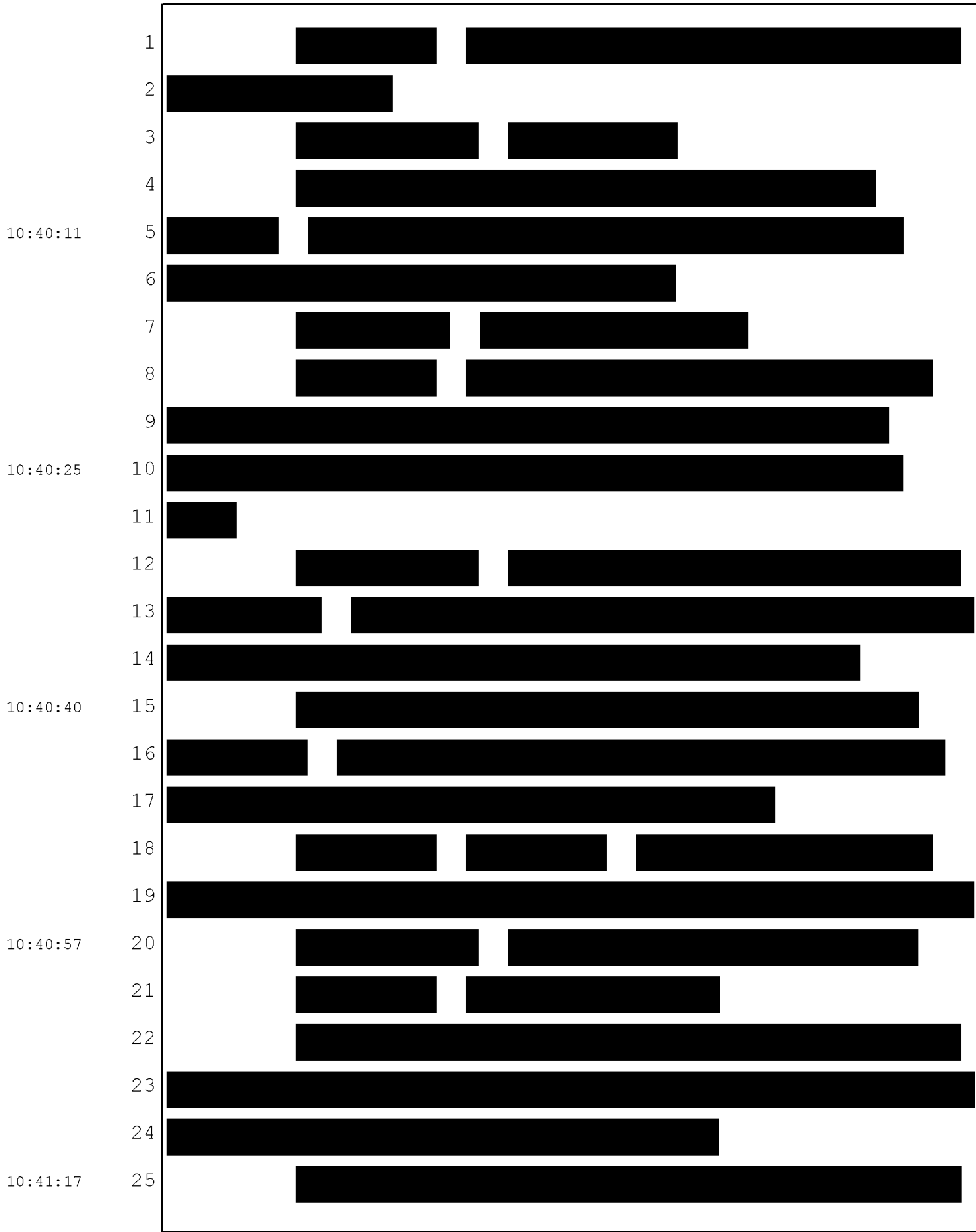


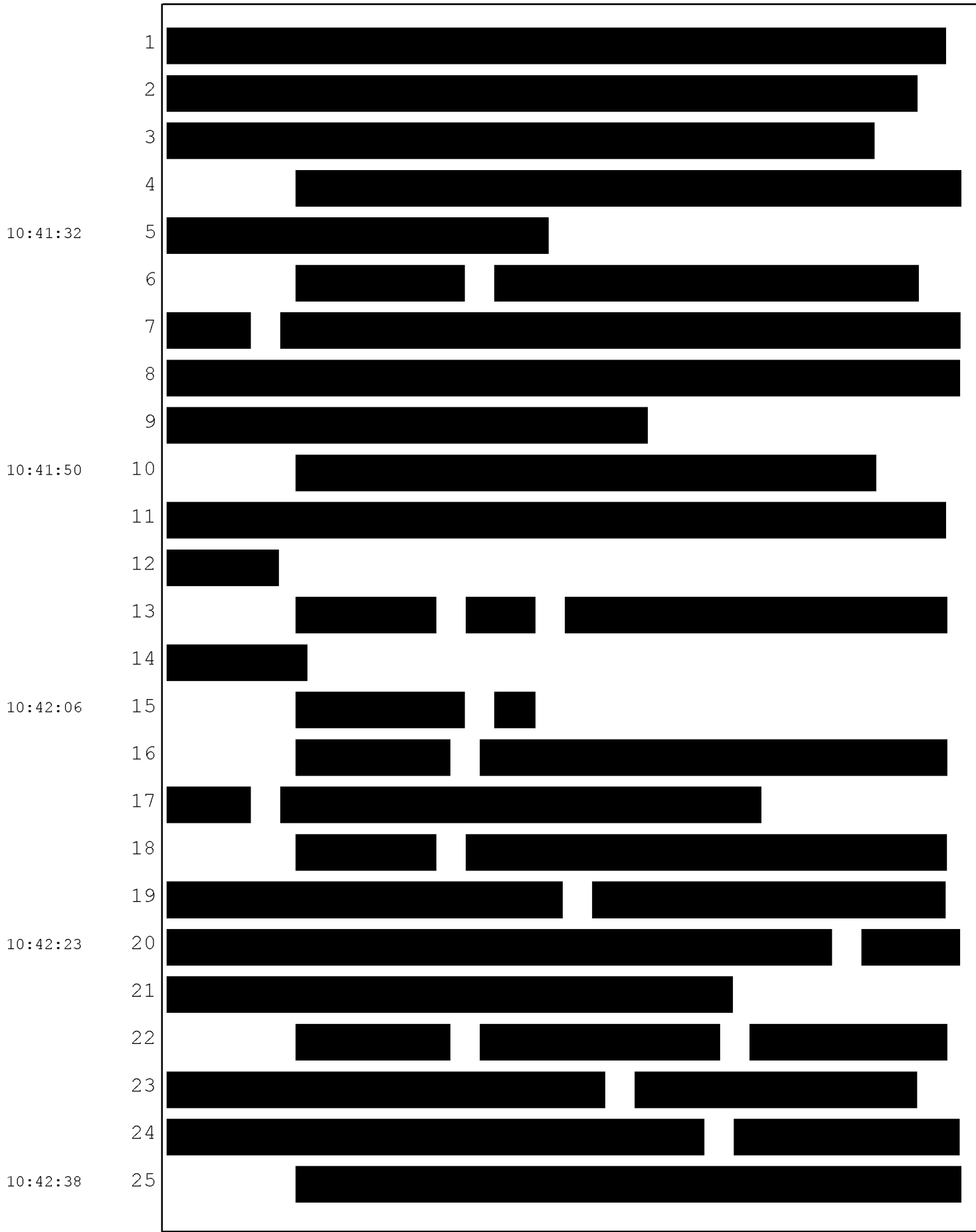


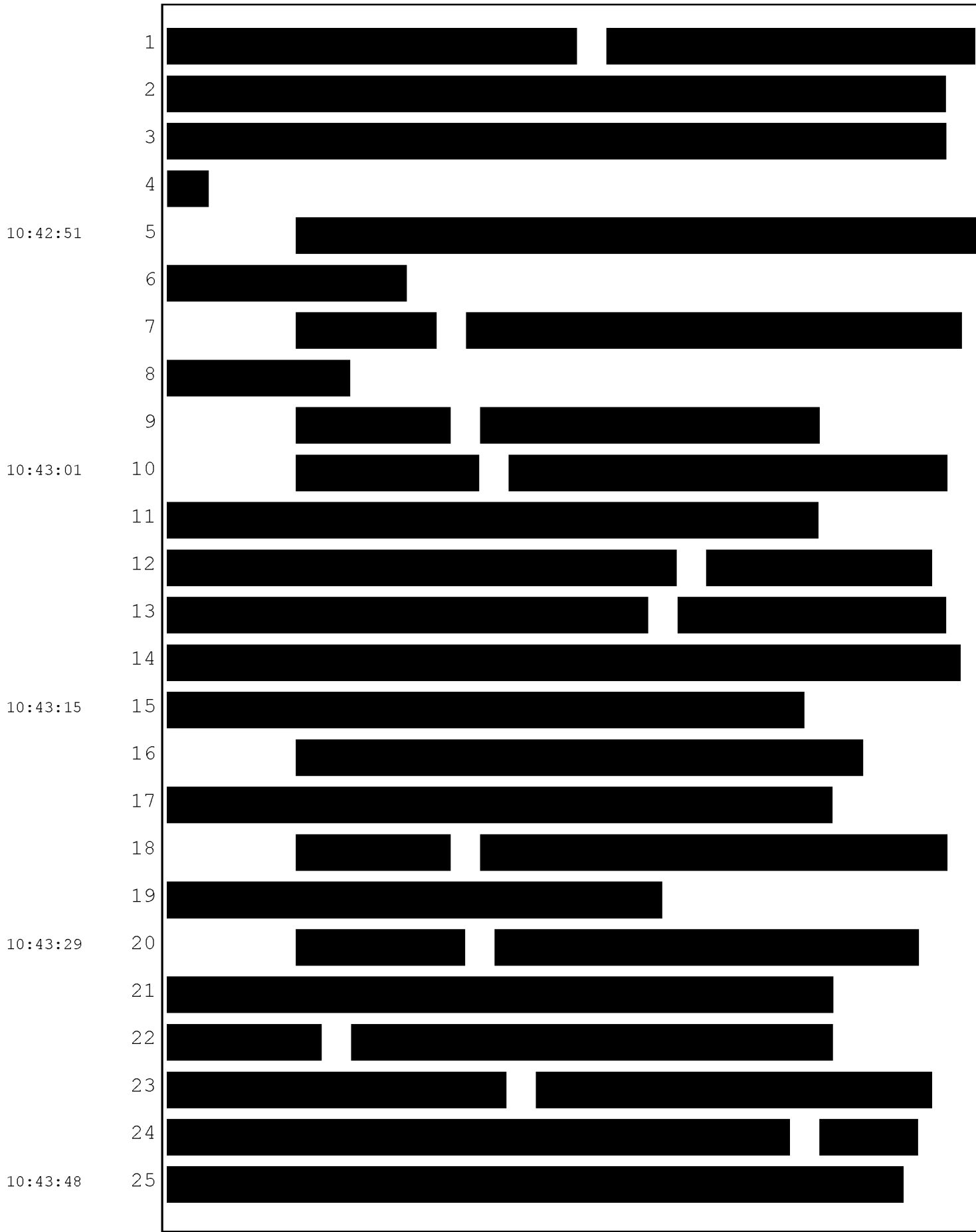












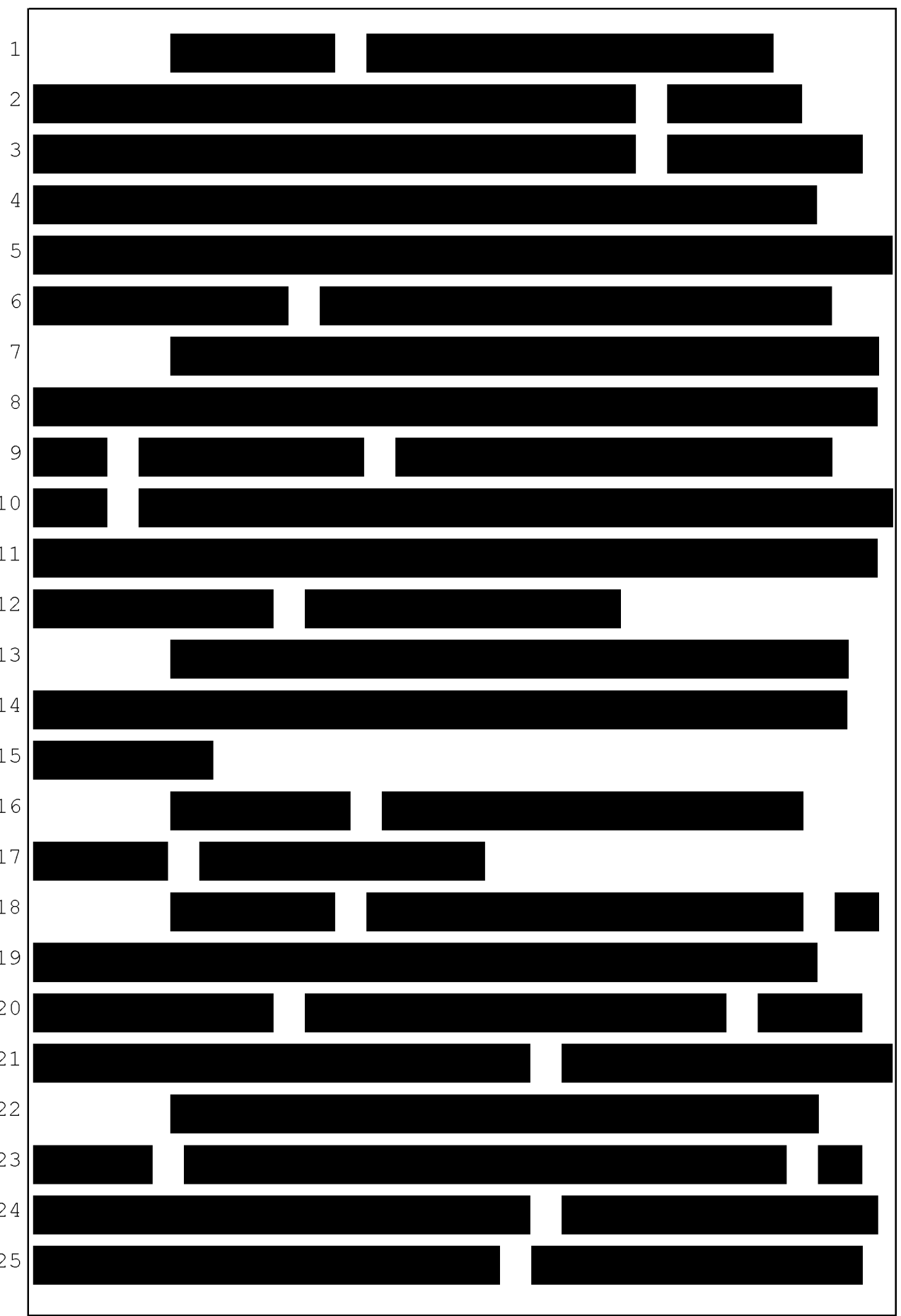
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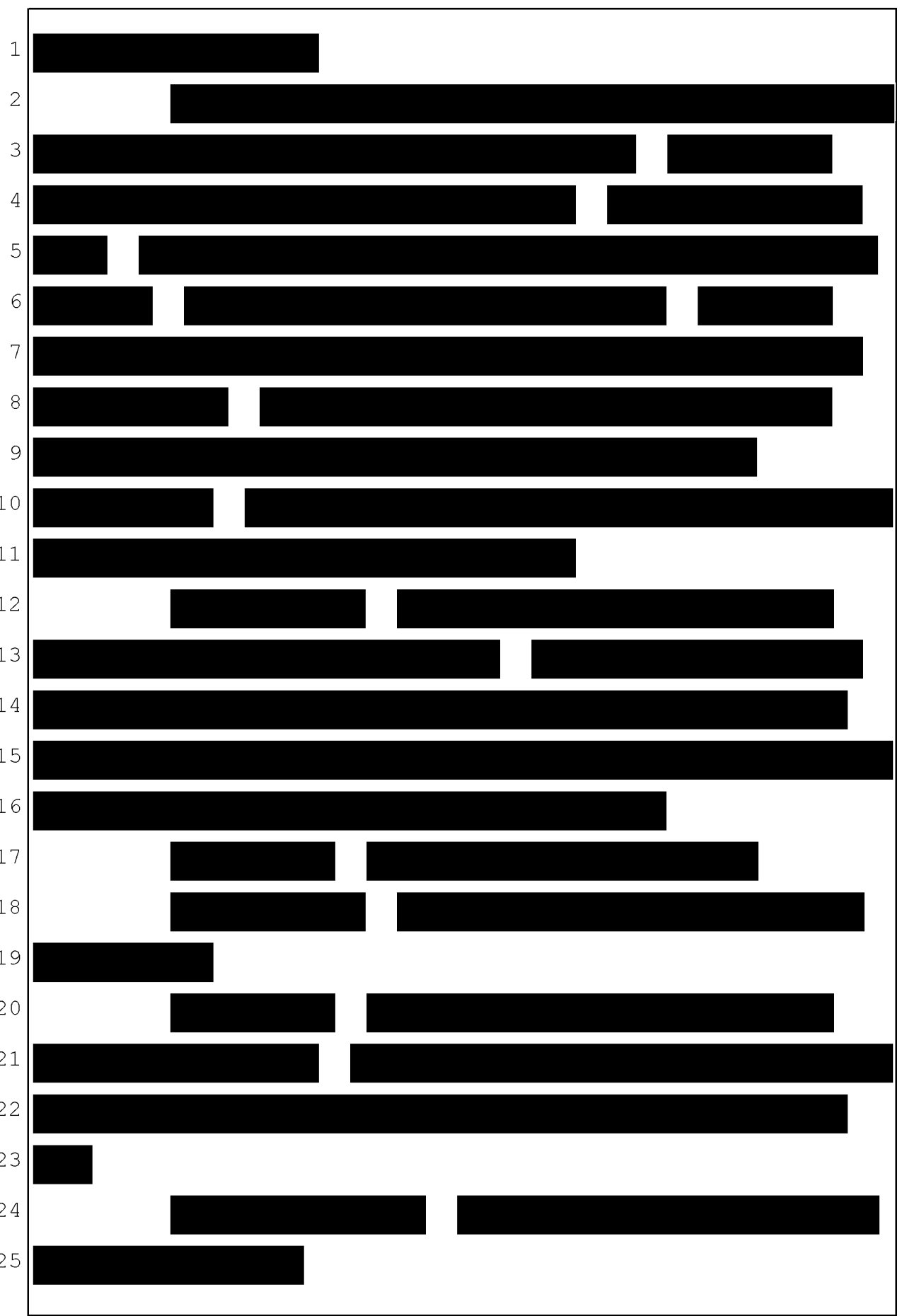
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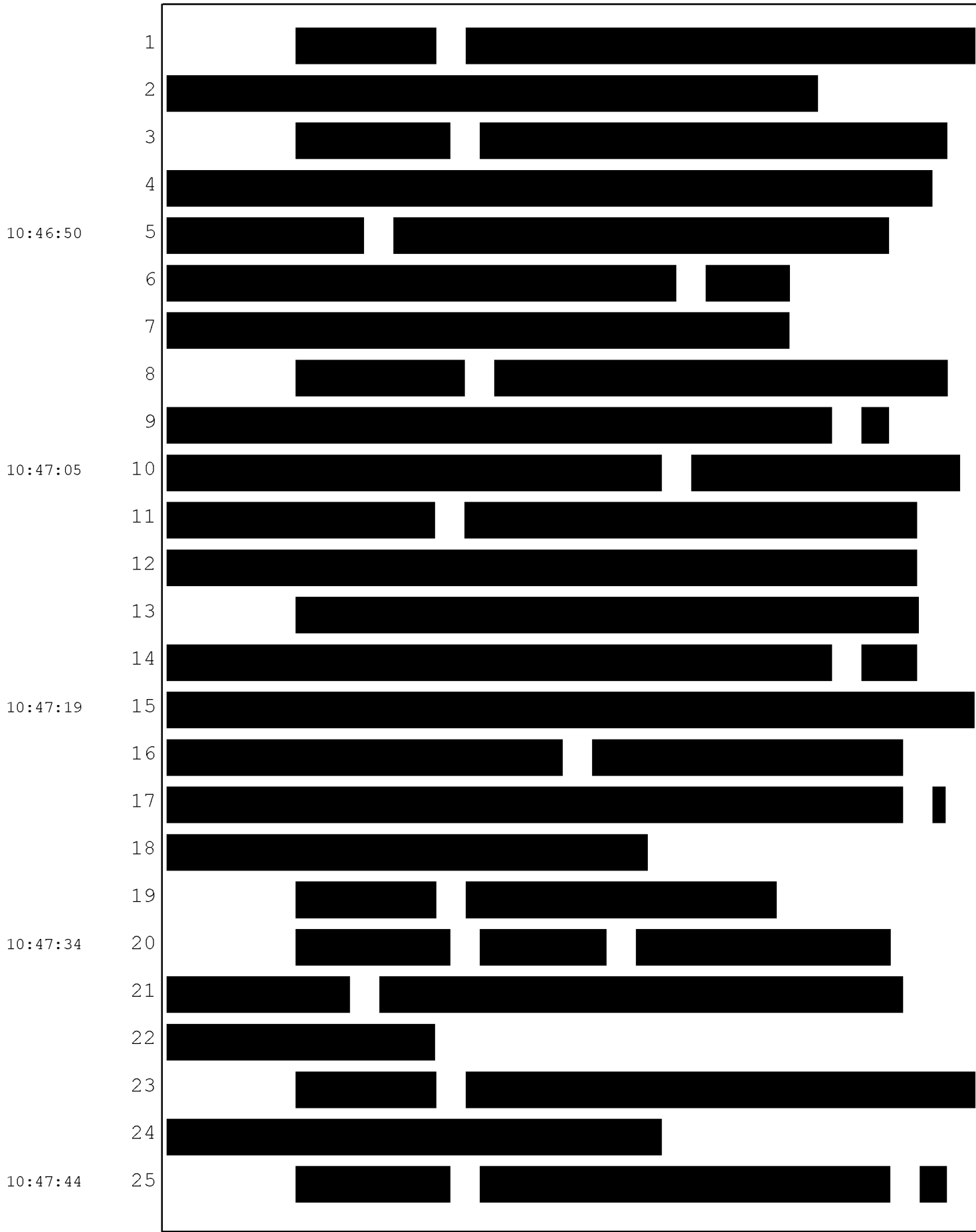
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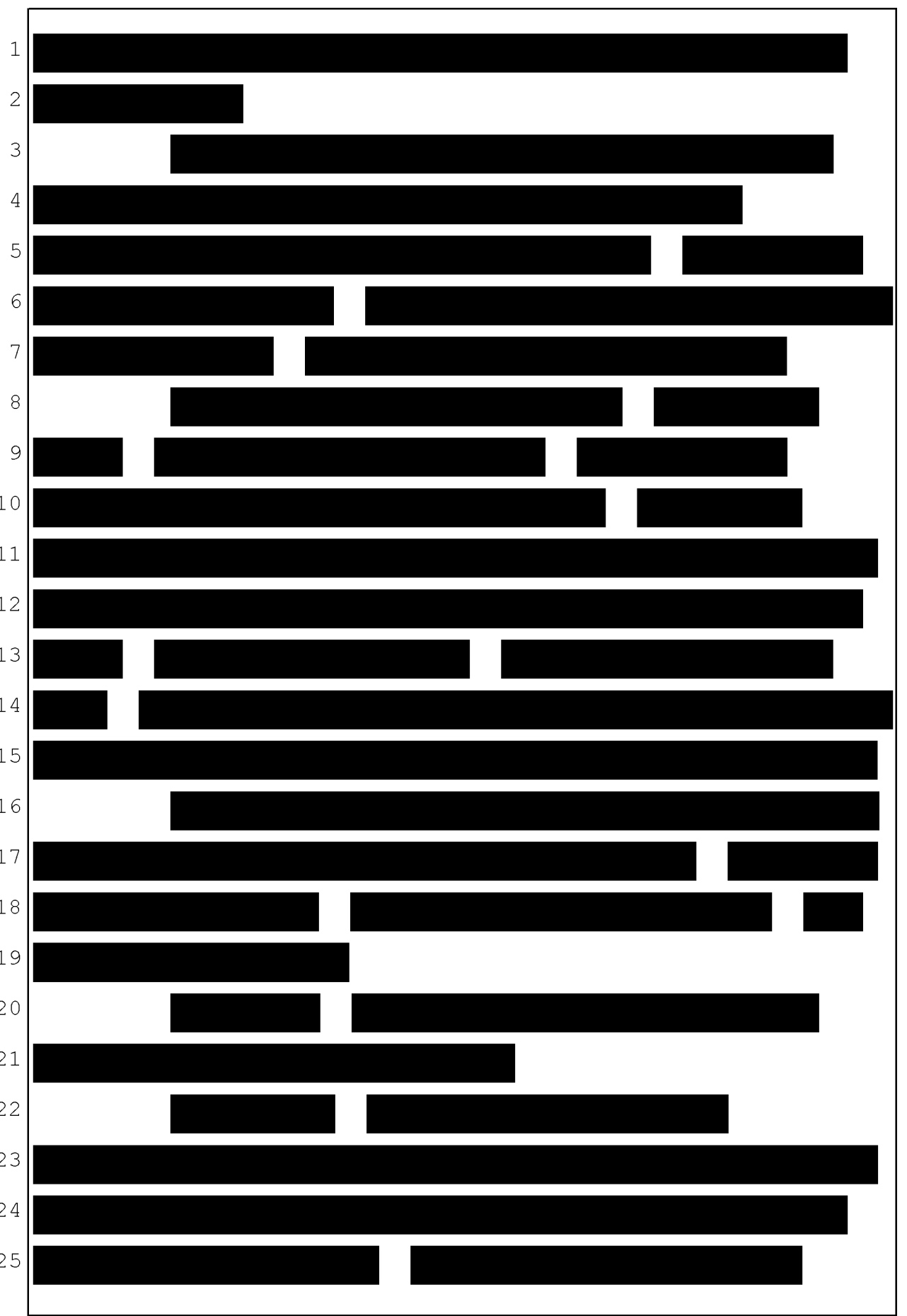
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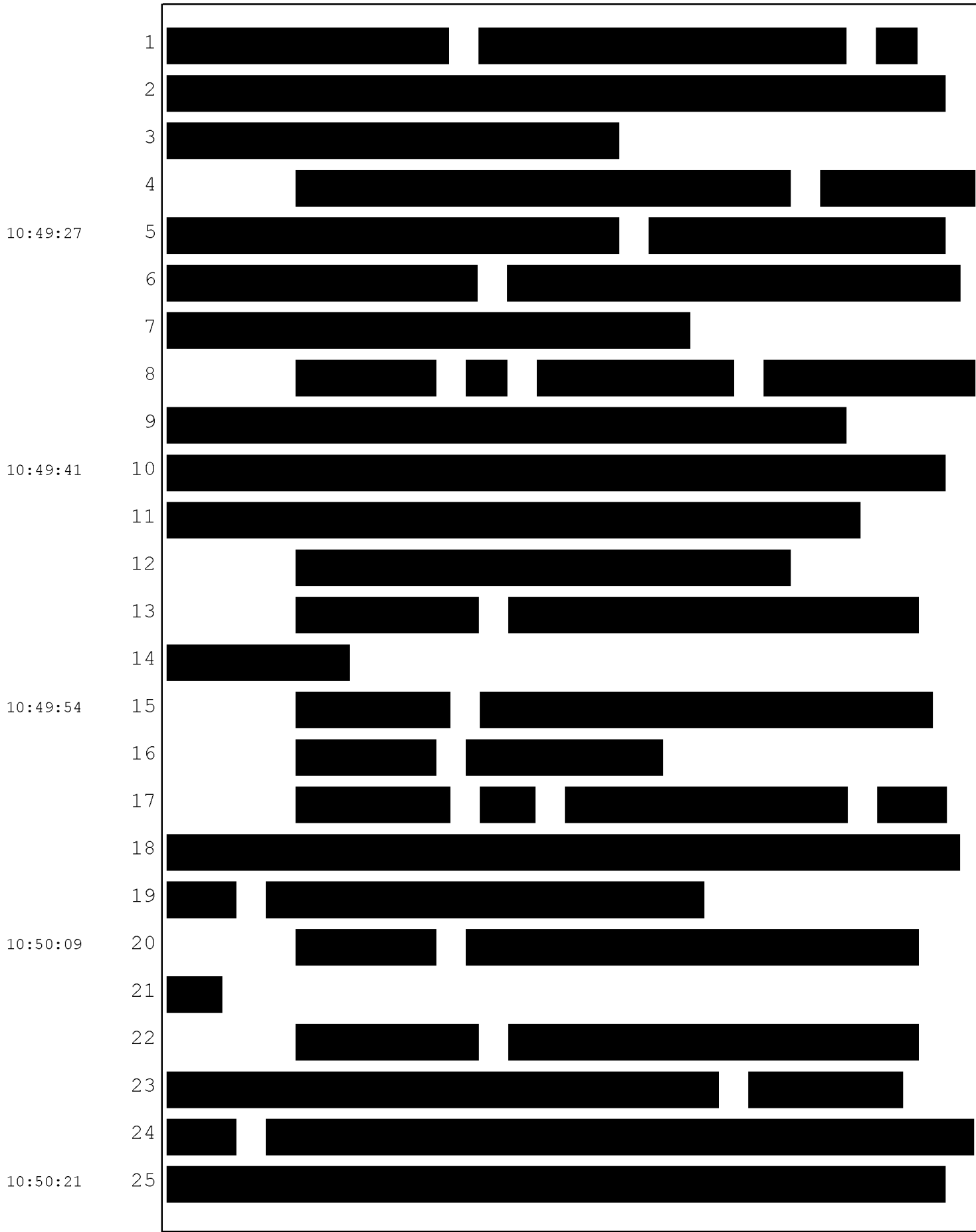
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(End sidebar.)

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 Issues Regarding Confounding and Exposure Misclassification in Epidemiological Studies of Occupational Exposure."

10:51:26

Do you see that, Doctor?

A. Yes.

10:51:37

Q. Just before the little break there, we talked

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

10:51:52

5 Do you see that, Doctor?

6 A. Yes.

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

9 What is confounding?

10:51:56

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

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

10:52:21

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

10:52:37

20 Q. That's correct.

21 A. So confounding is that there is a third factor  
22 which explains the association between the two, which is  
23 not necessarily causal, so -- and to have confounding,  
24 the -- both the exposure and the outcome have to be  
25 associated with this third factor, whatever that might

10:52:56

1 be.

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

10:53:12

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

10:53:45

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

13           So in that association -- again, if you did  
14 yellow fingers as the exposure and lung cancer as the  
15 outcome, you would find a statistical association between  
16 yellow fingers and lung cancer. But the confounder would  
17 be tobacco.

10:54:04

18           Tobacco is associated with both getting yellow  
19 fingers and tobacco is associated with getting lung  
20 cancer. So that's confounding. You're confounded by  
21 tobacco, so --

10:54:21

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

10:54:33

25           A. Right.

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

3 A. No.

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

10:54:47

5 A. Well, I would -- so I would say I know it's not  
6 a confounder because it's biologically implausible,  
7 but -- so that's how I would say it.

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

10:55:03

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

13 Q. But, Doctor, if you control for yellow fingers,  
14 right, it would actually eliminate the association with  
15 smoking; right?

10:55:16

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

18 Q. So before you control for any confounder, you  
19 have to have two parts; right? It has to be associated  
20 with the exposure. So yellow fingers, that's associated  
21 with smoking; right?

10:55:23

22 A. Uh-huh.

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

24 A. So if two things are really highly correlated  
25 with each other, it can be hard to sometimes tell them

10:55:33

1 apart. It's not always easy.

2           So that's why God gave us brains. We're  
3 supposed to actually think when we do studies and use  
4 some sense of logic. And this was -- I would say how to  
10:55:50 5 interpret studies, how to interpret associations, how to  
6 interpret causality, is not purely a statistical function  
7 but we're supposed to use our judgment and our intuition,  
8 and our -- and, again, not -- not -- and that's subject  
9 to judgment and to thinking and to -- and to the rules of  
10:56:16 10 any other exercise in human behavior of the thought  
11 process. So we have to think about it and do other  
12 studies and, you know, see how it works.

13           Q. All right. Let's talk about exposure  
14 misclassification. What is that in the context of  
10:56:35 15 epidemiology?

16           A. So, again, if we go back to our little  
17 assessment here, to the degree that you have error in the  
18 measurement of either the outcome or the exposure, that's  
19 obviously going to cause a problem in terms of how you  
10:56:56 20 assess the risk estimate or how you assess the  
21 association between the two.

22           Most cancer epidemiologic studies, we're pretty  
23 good in measuring the outcome. In other words, we know  
24 when someone's got cancer and when they don't have  
10:57:12 25 cancer.

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

10:57:22

5 If we were studying depression, we might have  
6 trouble in measuring with clarity or with validity or  
7 with precision the exposure outcome.

10:57:43

8 But in cancer, we usually know when someone's  
9 got cancer and when they don't have cancer. So in most  
10 cases in cancer epidemiology, the outcome is actually  
11 measured with a high degree of validity. We know when  
12 people have lymphoma, breast cancer, colon cancer,  
13 prostate cancer.

10:57:55

14 The exposure, on the other hand, depending on  
15 the exposure, it may be measured with a high degree of  
16 precision, a high degree of validity or not. It depends  
17 on how it's measured and what the specific exposure may  
18 or may not be.

10:58:08

19 To the degree that there's exposure  
20 misclassification, that creates an instability in our  
21 measurement of the -- of the risk estimate -- of the risk  
22 ratio as a relative risk.

10:58:27

23 Now, this is -- we're talking now about what I  
24 would call randomness classification. If this was a  
25 dietary study, for example, and I have to -- how much



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

10:58:44

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

10:59:02

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

10:59:21

15 Some people, the other way. This is random error.

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

10:59:37

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

10:59:55

24           So what a random error will do is just exposure  
25 misclassification. If we don't measure the exposure

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

11:00:10

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

8 Q. That's okay.

9 A. But what -- so random exposure

11:00:23

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

11:00:44

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

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

11:01:01

20 you may lose that relative risk estimate because of  
21 exposure misclassification -- randomness exposure  
22 misclassification, and you'll measure a 1 instead of a  
23 1.5. You'll lose it entirely because of exposure  
24 misclassification. And you don't need much exposure

11:01:16

25 misclassification to lose a relevant risk of 1.5.

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

3 A. Aaron Blair is the former head of something or  
4 other at the National Institute of Environmental Health  
11:01:38 5 Sciences. He's a hotshot guy in the -- the environmental  
6 and occupational epidemiologic -- I'm one of the leading  
7 scientists in this country in this area, and actually,  
8 he's a coauthor on many of the papers that are relevant  
9 to our discussion today.

11:01:54 10 Q. He also chaired the IARC program on glyphosate;  
11 right?

12 A. He chaired the -- I don't know if he chaired the  
13 whole IARC thing or he -- he certainly chaired the  
14 cancer -- yes, he chaired the epidemiology subcommittee.  
11:02:07 15 I don't know if he chaired the whole Working Group. But  
16 he chaired the cancer -- the epidemiology sub-committee.

17 Q. So in this study, they state -- they state:  
18 "Confounding and exposure misclassification are issues  
19 that concern epidemiologists because of their potential  
11:02:27 20 to bias results of studies and complicate  
21 interpretations."

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

24 A. Absolutely.

11:02:33 25 Q. "In occupational epidemiology, both are

1 routinely raised to argue that an observed result is  
2 either a false positive or a false negative finding.  
3 Although it is important to consider the potential for  
4 limitations of epidemiological investigations. Judgment  
11:02:50 5 regarding their importance should be based on their  
6 actual likelihood of occurrence."

7 Do you see that, Doctor?

8 A. Yes. So that's what I was saying before.  
9 Judgment is what I was saying before, which is basically  
11:03:01 10 saying that we're supposed to use our brains to think  
11 about how to interpret what we see.

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

11:03:12 15 THE COURT: Okay.

16 Q. BY MR. WISNER: All right. Well, so in this  
17 study, Dr. Blair and his colleagues, they go  
18 systematically through some of the science on confounding  
19 and misclassification exposure, and I want to look  
11:03:23 20 through their conclusions.

21 It says, "Conclusions. We believe that of the  
22 two major methodological issues raised in epidemiologic  
23 studies of occupational exposures, that is confounding  
24 and exposure misclassification, the latter is of far  
11:03:37 25 greater concern."

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

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

11:03:47 5           Q. Oh, sorry. Yes.

6           A. I think that's correct, that we don't measure  
7 exposure with great precision. When you ask someone how  
8 many are -- were you exposed to some exposure -- there's  
9 a high error rate in terms of -- it could be a random  
11:04:06 10 error rate, but there's a high error rate, like what I  
11 was saying before with broccoli. You just don't get it  
12 right. No one does.

13           Q. They go on to say, "It is rare to find  
14 substantial confounding in occupational studies or in  
11:04:20 15 other epidemiologic studies for that matter, even by risk  
16 factors that are strongly related to the outcome of  
17 interest. On the other hand, exposure misclassification  
18 probably occurs in nearly every epidemiological study.  
19 For nondifferential misclassification, the type of  
11:04:35 20 misclassification most likely in cohort studies, the  
21 direction of bias is largely predictable. That is a bias  
22 of relative risks towards the null."

23           Do you see that?

24           A. Yes. So that's exactly what I was saying  
11:04:50 25 before, that when you get random error in -- in measuring

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

11:05:13

5 Q. And you would agree, Doctor, with Dr. Blair and  
6 his colleagues that the most -- the thing to be most  
7 concerned with in evaluating the methodological issues of  
8 epidemiology, you're most concerned with exposure  
9 misclassification, more so than confounding, although you  
10 are concerned with both?

11:05:33

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

13 Q. Okay. And you agree with that?

14 A. Yeah.

11:05:39

15 Q. Okay. Great.

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

18 THE COURT: All right. Ladies and Gentlemen,  
19 we'll take the morning recess now. We'll be in recess  
20 until 11:20 on the clock. Please remember not to discuss  
21 the case, and we'll resume again at 11:20. Thank you.

11:05:47

22 (Sidebar.)

23 [REDACTED]

24 [REDACTED]

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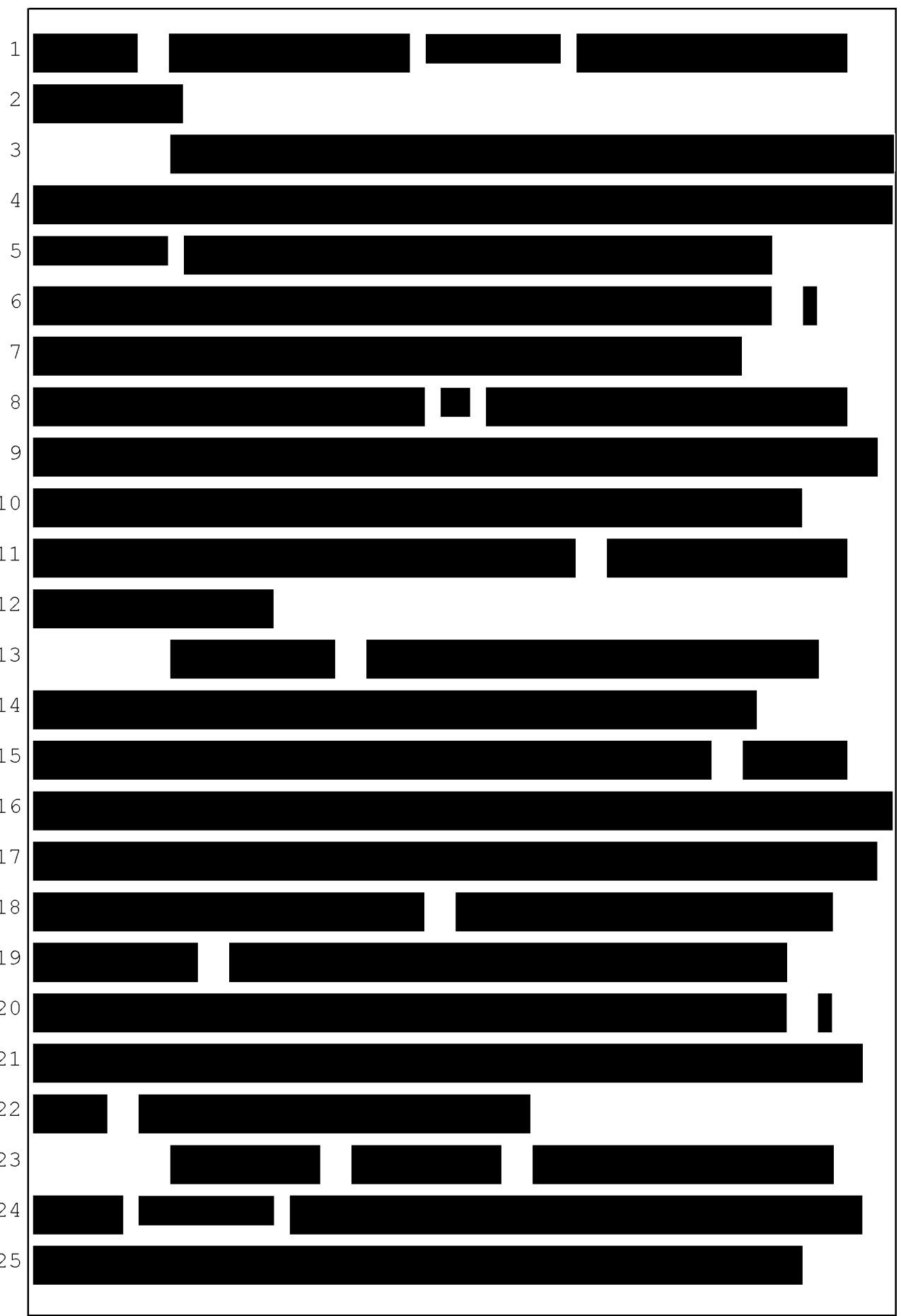
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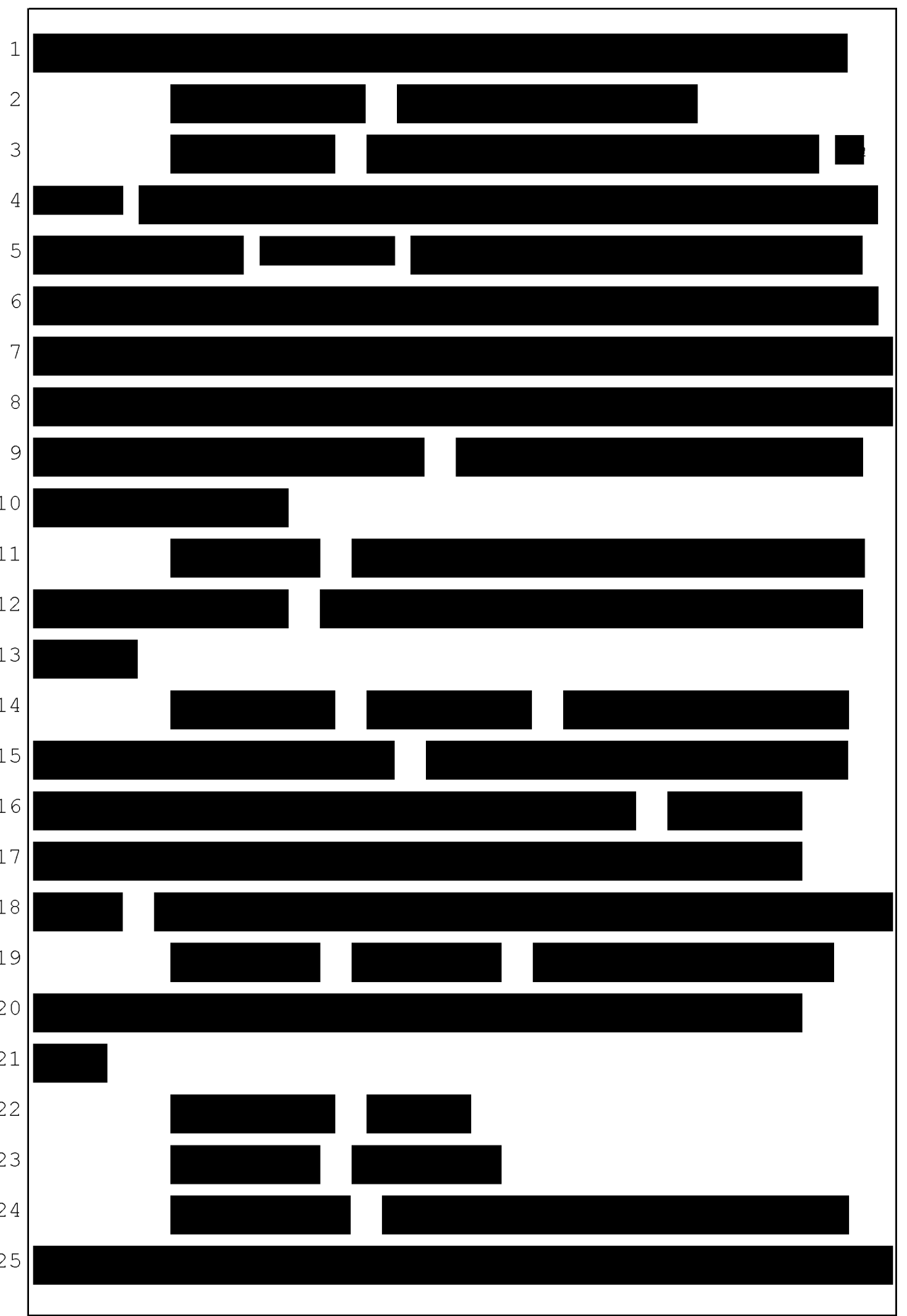
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8 (Recess.)

11:21:25

9 THE COURT: Welcome back, Ladies and Gentlemen.  
10 Dr. Neugut remains under oath, and, Mr. Wisner,  
11 you may continue.

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

14 THE COURT: Any objection?

11:21:36

15 MR. LOMBARDI: No objection, your Honor.

16 THE COURT: Very well.

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

11:23:06

19 During opening statements, Mr. Lombardi showed  
20 the jury an image that had a couple of IARC probable  
21 carcinogens, one included a -- very hot beverages and  
22 nightshift workers. Let's back up.

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

11:23:24

25 A. So in Northern Iran, there's the highest rates

1 of esophageal cancer in the world by far, and no one knew  
2 why, and it turned out in studies that the Iranians  
3 there -- I'm not making any political statements. But  
4 the Iranians there drink extraordinarily hot tea, and  
11:23:52 5 studies have shown that -- that the tea can -- when it's  
6 drunk, can burn the esophagus going down. We're talking  
7 about -- we're not talking about the same level of heat  
8 that you drink with the Queen of England. We're talking  
9 about a level of heat that's greater than, say, 150  
11:24:14 10 degrees, 160 degrees Fahrenheit.

11 The normal level of tea that, let's say, you and  
12 I drink, I guess, or the average American would drink in  
13 tea or coffee is about 130, 140 degrees, but they drink  
14 it at about 160, 170 degrees, and they drink it right  
11:24:31 15 away after making it. And it shows a high correlation  
16 between those who drink this hot level of tea -- and  
17 particularly they drink it right after they make it, so  
18 they don't wait too long after it's drunk. And they  
19 drink tea -- that's their main beverage.

11:24:45 20 So it scalds -- it scars, basically, their  
21 esophagus, and scarring is known to cause squamous cell  
22 carcinoma of the esophagus, so that's what IARC is  
23 talking about, that kind of -- a high level of esophageal  
24 carcinoma, which applies to hot tea.

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

1 China, where they also drink very hot tea. It's not a  
2 question in the world about -- no pun intended -- about  
3 this being a carcinogenic phenomenon. We're not talking  
4 about, again, the tea you get in Starbucks or something  
11:25:23 5 like that or that you might make in your home. We're  
6 talking about really hot tea.

7 Q. What about nightshift workers?

8 A. Nightshift workers are known to be at a high  
9 risk for all sorts of medical issues, because they're  
11:25:38 10 circadian rhythm has been disrupted. Their hormonal  
11 balance of -- when you -- there's twice in a day when  
12 your hormones are, sort of, up. I mean, you might  
13 recognize it by the fact that you wake up in the morning  
14 before your alarm clock does because of your circadian  
11:25:57 15 rhythm.

16 So if you're going to be, you know, getting up  
17 at night and doing nightshift instead of the normal --  
18 like with a rooster every day, that shifts all your  
19 hormonal balances, and that relates to getting different  
11:26:09 20 kinds of cancer that are -- if you're permanently or  
21 usually on nightshift work, then it changes your hormonal  
22 balances in such a way as to lead to various cancers.  
23 I'm not an expert on that, to be honest, but it's  
24 certainly totally accepted within the cancer epidemiology  
11:26:29 25 community that this kind of nightshift work causes cancer

1 of various sorts.

2 Q. And here's another slide that was shown to the  
3 jury, now that that's working. It says that, "Of the  
4 1,003 classifications made by IARC, only one is probably  
5 not carcinogenic."

11:26:47

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

8 A. No. That's idiotic.

9 Q. Why is that?

11:26:56

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

11:27:19

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

17 Q. Unclassifiable, not enough data.

18 A. Essentially, which would classify as probably  
19 50 percent or more of the chemicals or exposures that  
20 were looked at, which basically says not likely to be  
21 associated with cancer, which is more the way we talk in  
22 real everyday science and in epidemiology.

11:27:33

23 So of the thousand-odd chemicals or exposures  
24 that have been looked at, most of them are classified as  
25 probably not -- as not carcinogenic, so that's more

11:27:54

1 reflective of the reality of the exposures.

2           Only about 10 to 15 percent of the exposures  
3 that IARC has looked at have been classified as 1 --  
4 Level 1 or Level 2A. Glyphosate is 2A -- as Level 1,  
11:28:19 5 which is a definite carcinogen, or 2A, which is a  
6 probable carcinogen. It's only about 10 to 15 percent of  
7 the total are in those that -- are in those two highest  
8 categories.

9           Q. Now, Doctor, please turn in your binder to  
11:28:31 10 Exhibit 793.

11           Are you there, sir?

12           A. Yes.

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

11:28:56 15           Do you see that?

16           A. Yes.

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

11:29:02 20           A. Yes.

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

24           A. Okay.

11:29:29 25           Q. Okay. And there's a section that reads: "IARC

1 evaluates only agents that have some evidence of  
2 carcinogenicity."

3 Do you see that? Page 7.

4 A. Give me a sec.

11:29:45 5 Q. Middle of the page.

6 A. Yes.

7 Q. Okay. I'm just going to read a few of these  
8 bullet points and ask you about them. The first one  
9 says -- actually, Doctor, just to back up. If you look  
11:29:56 10 to the first page on this, I just want to read what this  
11 is.

12 It says in the first paragraph, "Since the  
13 evaluation of glyphosate by the IARC Monograph's program  
14 in March 2015, the agency has been subjected to  
11:30:12 15 unprecedented coordinated efforts to undermine the  
16 evaluation, the program and the organization. These  
17 efforts have deliberately and repeatedly misrepresented  
18 the agency's work."

19 Do you see that?

11:30:23 20 A. Yes.

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

24 A. Yes.

11:30:32 25 Q. Seems to be specifically criticisms based on

1 their classification of glyphosate.

2 Do you see that?

3 A. Yes.

4 Q. All right. So turning back to page 7, the first  
11:30:44 5 bullet point under that heading reads: "Some critics say  
6 the Monograph's program finds," quote, "everything causes  
7 cancer," end quote, "because of nearly 1,000 agents  
8 evaluated, only one has been categorized in Group 4,  
9 quote, "probably not carcinogenic to humans. The  
11:31:01 10 criticism is misleading, because the Monographs do not  
11 select at random the agents evaluated for  
12 carcinogenicity. Instead, in the interest of efficiency  
13 and according to the preamble of -- to the Monographs,"  
14 quote, "agents are selected for review on the basis of  
11:31:16 15 two main criteria, A, there's evidence of human exposure,  
16 and, B, there is some evidence or suspicion of  
17 carcinogenicity."

18 Do you see that?

19 A. Yes.

11:31:25 20 Q. Now, Doctor, you mentioned earlier that  
21 sometimes when you want to look to see if a compound is  
22 carcinogenic, IARC just hasn't looked at it. Do you  
23 recall that?

24 A. Yes.

11:31:36 25 Q. So what is this paragraph telling you about the

1 way IARC selects chemicals for review?

2           A. IARC has a process by which it solicits  
3 recommendations from general scientific and other  
4 communities to decide what agents should be evaluated by  
11:31:53 5 IARC. They don't want to waste their resources and time  
6 on evaluating everything in the world. So they only  
7 review things that have some prior evidence or some  
8 suggestion that they may be carcinogenic.

9           So by the time they're already being evaluated  
11:32:13 10 by IARC, they're already a cut above in terms of the  
11 likelihood that they're going to be carcinogenic. So if  
12 you say that half or not, or -- then you've already taken  
13 out like you're -- the pyramidal scale -- you're already  
14 in the upper part of the pyramid, in terms of the  
11:32:31 15 likelihood that they're going to be carcinogenic.

16           And as it says a paragraph or two later,  
17 "Despite this careful selection of agents, in reality,  
18 around half of the Monograph's evaluations resulted in  
19 agents being classified in Group 3" -- that's what I was  
11:32:46 20 referring to early -- "not classifiable as to its  
21 carcinogenicity in humans."

22           Which is really where the ones that are not --  
23 where the IARC Working Groups decide that they're really  
24 not truly carcinogenic. They fall into -- you know, it  
11:33:03 25 would be very difficult to say truly not -- definitely



1 not carcinogenic or probably not carcinogenic.

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

3 I want to draw your attention to another section  
4 that also came up, incidentally, in Mr. Lombardi's  
11:33:18 5 opening statement. If you turn to page 8.

6 A. Page 8 in this document?

7 Q. Yes, that's right.

8 It reads: "Monograph evaluations take account  
9 of," quote, "real-world exposures by evaluations of  
11:33:32 10 epidemiological studies."

11 Do you see that?

12 A. Yes.

13 Q. All right. It reads: "A charge level at the  
14 Monographs is that evaluations are divorced from the real  
11:33:39 15 world, i.e., are made without taking account of realistic  
16 human exposures. However, epidemiological studies are an  
17 essential part of Monograph evaluations and by definition  
18 deal with people exposed in daily life, including at  
19 work. The studies frequently consider the gradient of  
11:33:57 20 risk observed with different levels of exposure. One  
21 part of the Monograph evaluation is specifically  
22 dedicated to describing the circumstances under which  
23 human exposure occurs and at what levels."

24 And then the last thing it says, "In light of  
11:34:14 25 the occurring," quote, "real-world human exposures,

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

3           Now, Doctor, is it your understanding, as an  
4 epidemiologist who relies on IARC routinely in your  
11:34:29 5 practice, when IARC classifies something as a probable  
6 human carcinogen, does that mean it's not a real-world  
7 carcinogen?

8           A. Of course it's a real-world carcinogen. I mean,  
9 it would be nice, speaking as a scientist, to be able to  
11:34:43 10 do these studies the same way we do with animals and take  
11 25 or 30 people and put them in a room and give them the  
12 maximum tolerated dose and see what happens, but I'm not  
13 allowed to do that.

14           So in essence, we use the -- obviously, the  
11:34:58 15 epidemiologic studies are relying on how people are  
16 really exposed in day-to-day life. All the studies that  
17 we're going to talk about or that we hear are, basically,  
18 asking farmers or agricultural workers or whomever how  
19 much they've been exposed to. What could be more real  
11:35:16 20 life? That's exactly what people are exposed to. That's  
21 what epidemiologic studies are.

22           Q. Now, if you look at the bottom of this page, the  
23 last bullet point, it reads: "In practice, by far the  
24 most frequent change in classification after  
11:35:29 25 re-evaluation is that the agent goes into a higher group,

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

11:35:48

6 Can you explain what you understand that to  
7 mean?

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

11:35:59

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

11:36:20

11:36:40

11:36:58

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

4 Q. Now, Doctor, the reason why IARC classified it  
11:37:12 5 in 2A as opposed to 1 was because the epidemiological  
6 study was, in IARC's view, limited. Do you recall that?

7 A. Yes.

8 Q. Have there been examples in the past where  
9 something was originally classified by IARC as 2A because  
11:37:30 10 of limited epidemiology, but as time went on, they  
11 developed more epidemiology because people are using it  
12 in the world and they upped it to 1?

13 A. So an example of that is formaldehyde.  
14 Formaldehyde is a chemical that's used in the  
11:37:43 15 occupational setting for a variety of -- in construction,  
16 and it's used in the undertaking -- you know, in  
17 embalming and things like that.

18 So that was classified as a 2A originally, and  
19 based mainly -- just like glyphosate, based heavily on  
11:38:03 20 the toxicological evidence and with limited epidemiology,  
21 so it was initially classified as 2A, and then some years  
22 later, it had a re-evaluation when there was more  
23 epidemiology evidence, more studies had been done, and it  
24 got reclassified, and it's now classified as a definite  
11:38:26 25 carcinogen, as a Class 1 carcinogen by IARC.

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

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

19 Do you see that, Doctor?

20 A. Yes, sir.

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

23 A. So what --

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

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

3 THE COURT: All right. Sustained.

4 Please ask a different question.

11:39:56 5 Q. BY MR. WISNER: What is your understanding of  
6 IARC's role in epidemiology and cancer research?

7 A. IARC, as I said at the beginning of my  
8 testimony, to me is the number one arbiter in the world  
9 of whether something is actually carcinogenic and what  
11:40:14 10 the level of probability is that it is a carcinogen or  
11 not. What to do about it or what public policy should be  
12 about how to handle that information is for others to  
13 decide. I don't think IARC has that as a goal or an  
14 intent. It's for other agencies to use the  
11:40:35 15 information -- that information as they see fit.

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

19 Do you see that?

11:40:53 20 A. Yes.

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

24 A. Yes.

11:41:03 25 Q. And in this section, the director is discussing

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

3 A. Yes.

4 Q. Okay. And at the very end of it he quotes  
11:41:18 5 testimony from Dr. Blair, who was a chief investigator of  
6 the AHS, as well as the chair of the IARC Monograph.

7 Do you see that?

8 A. Yes.

9 Q. And to the best of your understanding, did IARC  
11:41:31 10 change its classification following the publication of  
11 the Andreotti paper in 2017?

12 A. No.

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

11:41:50 15 MR. LOMBARDI: Would you repeat the number,  
16 please?

17 MR. WISNER: 284.

18 MR. LOMBARDI: Thank you.

19 THE WITNESS: Okay.

11:42:07 20 Q. BY MR. WISNER: You've got it?

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

24 Do you see that?

11:42:18 25 A. Yes.

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

3 A. 2015.

11:42:32

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

6 A. Yes.

7 MR. WISNER: Permission to publish, your Honor?

8 THE COURT: Any objection?

9 MR. LOMBARDI: No objection, your Honor.

11:42:40

10 THE COURT: Very well. You may proceed.

11 Q. BY MR. WISNER: So this is the article, Doctor.

12 And what they're doing in this article, and correct me if

13 I'm wrong, is they're reviewing has IARC been correct and

14 effective in the last 40 years since its existence; is

11:42:56

15 that right?

16 A. Yes.

17 Q. All right. And if we look here at the list of

18 authors, there are over 120 authors on this publication;

19 isn't that true?

11:43:07

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

22 Q. Okay. And this would be consistent with your

23 testimony earlier that IARC is widely respected within

24 the academic community; right?

11:43:18

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



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

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

11:43:31

5 A. Yes.

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

10 A. Yes.

11 Q. It says, "Disagreement with the conclusions in  
12 an IARC Monograph for an individual agent is not evidence  
13 for a failed or biased approach. Some disagreement about  
14 the carcinogenic hazard of important agents seems  
15 inherent to the scientific enterprise and is unavoidable  
16 at early stages of hazard identification where IARC  
17 usually operates."

11:43:59

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

11:44:20

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

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

11:44:36

25 Substances now universally recognized as human

1 carcinogens, for example tobacco or asbestos, at one time  
2 went through a quite lengthy period of contentious  
3 debate. Any process in theory can be improved with fair  
4 and conservative criticism. Appropriate reviews may take  
11:44:56 5 place from time to time, and we would support continued  
6 review and improvement of the IARC processes. However,  
7 as a group of international scientists, we have looked  
8 carefully at the recent charges of flaws and bias in the  
9 hazard evaluations by IARC Working Groups, and we have  
11:45:13 10 concluded that the recent criticisms are unfair and  
11 unconstructive."

12 Do you see that?

13 A. Yes.

14 Q. Do you agree with that?

15 A. Yes.

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

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

19 Q. I -- just for my own education, Doctor, have you  
11:45:28 20 ever seen 125 scientists agree on anything?

21 A. I suppose, but not too often.

22 Q. Okay. All right. Let's move off of IARC.

23 Let's go into some epidemiology here. I don't want to  
24 spend too much time on it. We had a chance to talk to

11:45:49 25 Dr. Portier a bit about some of the studies, but I would

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

3 A. Yes.

4 MR. WISNER: Okay. Permission to publish?

11:46:00

5 THE COURT: Any objection?

6 MR. LOMBARDI: No objection, your Honor.

7 THE COURT: Very well.

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

11:46:27

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

14 THE COURT: Yes, you may step down.

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

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

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

11:47:11

11:47:28

1 1 means there's no association.

2           And these are one, two, three, four, five -- six  
3 studies. These are all case-control studies, like we  
4 alluded to earlier. Case-control, meaning that you  
11:47:44 5 started with people who had lymphoma, and controls,  
6 people who did not have the lymphoma.

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

9           A. Oh, I'm sorry. Yes.

11:48:01 10           And then looked at their exposure to glyphosate  
11 or their association with glyphosate. And if your risk  
12 ratio is above 1, that means that you have an increased  
13 risk -- an association of having an association between  
14 the exposure and the outcome. If it's below 1, it means  
11:48:22 15 that there's a protective effect, that, in fact,  
16 glyphosate -- theoretically, that glyphosate would  
17 actually protect you from lymphoma, if you're -- if you  
18 measure 0.8 or 0.7. It means you have a 30-percent lower  
19 risk of having lymphoma if you were exposed to  
11:48:44 20 glyphosate.

21           Theoretically, if everything was truly random,  
22 according to the null hypothesis, like we said -- like  
23 things should be, if it was truly random between  
24 glyphosate and non-Hodgkin's lymphoma, then these studies  
11:49:02 25 should be randomly distributed around 1. Half should be

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

3           And if you look, all of them are above 1. All  
4 of them. That's a phenomenon referred to in causal  
11:49:21 5 epidemiology as consistency. They're consistently  
6 elevated above 1. Whatever flaws, problems, issues we're  
7 all going to raise about these studies, one or the other,  
8 no studies are perfect, whatever things each study does,  
9 no study is identical. One does something. One -- each  
11:49:39 10 study does something differently. Each study -- but all  
11 the circumstances under which these six studies -- some  
12 of them control for different things, some of them are  
13 done in different populations. Some of them in  
14 Scandinavia. Some of them are in America. Some of them  
11:49:53 15 in Canada. Some of them are with farmers. Some of them  
16 are not.

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

11:50:13 20           Q. Meta RR.

21           A. Meta RR.

22           So you get what's called a meta RR. So what  
23 happens here is a study was done by some scientists, by  
24 Cheng and Delzell. And what they did was they put  
11:50:27 25 together the risk estimates. Basically, they combined

1 these six studies together to get a combined risk ratio  
2 so we would be able to see what the combined outcome was  
3 from the six case-control -- six studies. They're not  
4 all case-control, and see what the combined effect was of  
11:50:47 5 all of them.

6           And this is the combined effect, and the outcome  
7 is that the risk ratio was 1.3, meaning that there was a  
8 30-percent increased risk of non-Hodgkin's lymphoma in  
9 the context of glyphosate exposure, with a significant  
11:51:10 10 evaluation -- with a significant -- statistically  
11 significant 95-percent confidence interval, as it's  
12 called, so that's the combined effect of all.

13           Why do you need to combine them to get them? So  
14 the point is that all of them, or almost all of them,  
11:51:27 15 cross the 1 line, which means that none of them are  
16 statistically significant on their own. We like to have  
17 statistical significance if you can. We don't have that  
18 in most of the studies. Why don't we have statistical  
19 significance in most of the studies? That's because of  
11:51:45 20 what I alluded to earlier, which is we're dealing with a  
21 very uncommon outcome and a very uncommon exposure.

22           So to have a statistically significant outcome  
23 in any individual study is extremely difficult, but if  
24 you combine them -- and again, the fact that they're all  
11:51:59 25 consistently positive together makes them -- leads to a

1 statistically significant positive exposure.

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

11:52:23

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

11:52:38

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

11:52:53

11:53:06

16           Again, I'm not going to argue whether it's 1.3  
17 or 1.4, et cetera. That's not really the issue. The  
18 point that we should walk away with is that overall,  
19 there's a statistically significant increased risk in the  
20 1.3, 1.4, possibly 1.5, range. And that's basically what  
21 the case control studies are showing us.

11:53:29

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

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

1 kind of, explore some of the differences.

2 A. Did Dr. Portier show this?

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

11:53:39 5 A. Uh-huh. Yeah, uh-huh.

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

9 A. Uh-huh.

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

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

14 Q. Okay. So if you were not to do a log scale, you  
11:54:03 15 know, .1 would be right there and 10 would be -- who  
16 knows where; right?

17 A. Yeah, uh-huh.

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

11:54:10 20 A. That's correct.

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

24 A. It would look more dramatic if you did not  
11:54:21 25 have -- if you used a straightforward numerical scale.



1 Like a regular integer scale.

2 Q. Okay. Great.

3 Now, one of the things that I also want to bring  
4 up here is you mention one study here, the De Roos 2005  
11:54:39 5 study.

6 A. Yes.

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

9 A. That's correct.

11:54:45 10 Q. So there are -- for example, like in Eriksson,  
11 there's actually a 2.-something number that's  
12 statistically significant, that's not adjusted for other  
13 pesticides.

14 A. That's correct. So that would show in the dose  
11:54:59 15 response relationships, yes.

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

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

19 A. Again, that's what these -- the authors who made  
11:55:08 20 the Forest plot in the meta-analysis did. And I think  
21 that's -- again, we should be conservative and try not  
22 to, you know, overstate reality.

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

11:55:25 25 A. Yes.

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

3 A. Correct.

11:55:34

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

6 A. Yes. Yes.

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

9 A. Yes.

11:55:42

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

11:55:59

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

11:56:14

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

11:56:32

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

11:56:44

5           Q. All right. So we have on here De Roos 2005.  
6 And that's from the agricultural --

7           A. AHS study.

8           Q. Okay. Great.

11:56:55

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

13          A. Yes.

14          Q. You've reviewed them?

11:57:05

15          A. Yes.

16          Q. You considered it?

17          A. Yes.

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

11:57:10

20          A. No.

21          Q. Why?

11:57:29

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

1 were initially recruited in the 1993 to 1996 time frame.

2 What happened subsequent to 1993 to 1996 was

3 that there was a dramatic rise in the use of glyphosate

4 for various reasons, so that whatever was assessed in

11:57:59 5 terms of the initial exposure of glyphosate really became

6 almost useless information. I think we have a poster --

7 Q. A demonstrative. Do you want to show the jury

8 that?

9 A. If we can.

11:58:11 10 Q. Yeah. Absolutely.

11 MR. WISNER: Permission to publish 1032?

12 THE COURT: Any objection?

13 MR. LOMBARDI: No objection, your Honor.

14 THE COURT: Very well.

11:58:19 15 THE WITNESS: So, again, we're talking now about

16 a cohort study. So we're talking about people who were

17 exposed -- who were recruited in 19 -- 50,000 people

18 roughly. 50-something-thousand people who were exposed

19 between -- who were recruited between 1993 and 1996 or

11:58:40 20 so. And they were each given a questionnaire and asked

21 about their usage of glyphosate and other herbicides.

22 And then what happened is -- you can see here,

23 after 1996, there was a huge rise, almost tenfold, in the

24 use of glyphosate.

11:58:57 25 So, basically, if you relied on the level of

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

11:59:16

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

11:59:34

10 dramatically.

11 So basically what it says is you can't rely on  
12 the baseline questionnaire that was done in 1993, 1994,  
13 1995, because the glyphosate exposure has dramatically  
14 changed. So, okay, that's no one's fault.

11:59:48

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

18 So they went back and tried to reinterview  
19 everybody --

12:00:00

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

22 A. Right. So around 2005, they went back and tried  
23 to reinterview the 50,000-odd, not odd, but, you know,  
24 50,000-ish people who were in the study.

12:00:13

25 So now they run into what's a problem in cohort

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

4 Well, I don't have to tell you, if someone  
12:00:28 5 called -- this was by telephone. I don't know what you  
6 do. When someone calls me on the telephone and wants to  
7 interview me, I hang up. Or I don't answer the phone in  
8 the first place.

9 So what happened was they called the 50,000  
12:00:43 10 people. Again, it's a little more than 50,000, but I'm  
11 just using rough numbers for our discussion. When they  
12 called the 50,000 people, they got a cooperation rate, if  
13 you want to call it that, a follow-up rate, of  
14 62 percent. So 38 percent of the people that they  
12:01:00 15 called -- I don't think anybody here will be surprised by  
16 that -- 38 percent of the people did not respond. Or  
17 they did not get follow-up from 38 percent of the  
18 cohorts.

19 Now they don't have information on 38 percent of  
12:01:12 20 the cohorts in terms of their subsequent follow-up in  
21 terms of glyphosate.

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

24 A. Yes.

12:01:20 25 Q. For example, let's say they had been -- between

1 1992 and 1997, they'd never used glyphosate.

2 A. Right.

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

12:01:32

5 A. Yeah. And if you didn't interview them, you  
6 wouldn't know anything about it.

7 Q. And then let's say it's even worse.

8 let's say -- 2000, they started using it for a couple of  
9 years. They died in 2004 from non-Hodgkin's lymphoma.

12:01:45

10 Okay?

11 A. Yeah.

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

14 A. Because they'd be dead.

12:01:54

15 Q. Yeah. So that person, for purposes of the  
16 study, then, would be considered unexposed, and the  
17 cancer would then be assigned to the unexposed group?

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

12:02:09

20 Yes, that's what you said.

21 Q. Yeah, that's what I meant.

22 A. Uh-huh.

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

12:02:18

25 Dr. Blair.

1 A. That's correct.

2 Q. So what happened?

3 A. So what happened is -- so, correctly, what they  
4 did was -- then you have several problems. So, again,  
12:02:26 5 this would not be a problem -- this would not be a  
6 problem if the risk ratio was high. If the relative risk  
7 was 10, like the tobacco and lung cancer -- again, these  
8 are errors that, kind of, would have been made -- instead  
9 of a relative risk of 10, maybe we'd have a relevant risk  
12:02:46 10 of 9 or a relative risk of 8 and -- who cares.

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

12:03:03 15 Q. Let's stop right there.

16 A. And there are several errors here.

17 Q. Let's stop right there.

18 MR. WISNER: Your Honor, it's probably a good  
19 time to take a break, so we can get a time check and make  
12:03:12 20 sure we don't run into the buzzer.

21 THE COURT: Okay. Great.

22 All right, Ladies and Gentlemen. Then we'll --  
23 we'll break now for the lunch recess. Please remember:  
24 Do not discuss the case, do not do any research. And  
12:03:23 25 we'll see you again at 1:30. Thank you.



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(Jury leaves courtroom.)  
(Time Noted: 12:03 p.m.)

1 REPORTER'S CERTIFICATE

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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.

<%signature%>  
Leslie Rockwood Rosas  
Certified Shorthand Reporter  
State of California  
Certificate No. 3462