## EHR Interfaces May Increase Likelihood of Errors

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Los Angeles — Electronic health records have been proposed as one way to reduce medical errors, but their design can contribute to errors as well, Melonie Nance, M.D., said at the annual meeting of the American Academy of Otolaryngology—Head and Neck Surgery Foundation.

"The way doctors work, and the way we think about patient problems and diseases

is often completely mismatched with the way things are presented in electronic records," Dr. Nance, a resident, said in an interview

Dr. Nance and her colleagues at the University of Pittsburgh analyzed two cases of preventable medical errors that occurred in part because of computer interface design. In neither case did the error lead to patient injury, thus both were "near misses."

In the first case, a resident reviewed the pathology report of an operative biopsy pri-

or to a composite resection and noted that the diagnosis was squamous cell carcinoma, but failed to recognize that the date of the biopsy was from the previous year.

In the electronic record used, multiple pathology reports were displayed on the same screen. Further, pathology and operative reports were stored in separate categories and were not linked, even though both reports resulted from the same procedure. The problem consisted of both time-line and data-fragmentation errors.

Rather than presenting critical data in a way that links related information, the electronic record in this case had recreated a problem seen with traditional paper files where information is stored by data type, Dr. Nance said.

Standardized time lines, unambiguous links between related information, and data organized by problem are all potential solutions to this problem, Dr. Nance said. For example, pathology reports of a head and neck cancer should be displayed with other information about the specific cancer, whereas reports of a liver biopsy should be linked to other information

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about the patient's liver disease.

In the second case, a patient was discharged in acute renal failure 30 minutes after the renal failure had been noted and documented by the critical care fellow. The fellow had entered the diagnosis

into the electronic record at the end of a lengthy note but had not communicated the information to the otolaryngology resident who discharged the patient. The error was discovered quickly and the patient was readmitted 2 hours later.

The primary problem in this case was that data entry was mistaken for thorough communication. Critical patient information was hidden from the discharging physician and the record contained excessive information.

Dr. Nance and her colleagues suggested that a severity scale could be used to bring attention to important information such as abnormal lab data. Copied-and-pasted notes, a strategy often used to generate complete documentation, could be marked with color coding, time stamps, or a notation similar to the "track changes" function on word processors.

# CHANGING THE WAY WE SEE RA IN RHEUMATOID ARTHRITIS B Cell Cytokine Production Autoantibody Production Autoantibody Production Intercultular Signaling Plasma Cells Plasma Cells Plasma Complexes RE Immune Complexes Amplification of Inflammation and Damaga

### Autoreactive B cells produce autoantibodies that may help drive the disease process in $RA^{3,5,10,11}$

B cells produce autoantibodies such as RF, anti-CCP, anti-GPI, and anti-RA33. RF immune complexes within the synovium may

- activate the complement system and stimulate an immune response<sup>3,10</sup>
- bind to, and activate, macrophages in the synovium<sup>11</sup>

Macrophages activated by immune complexes produce proinflammatory cytokines that perpetuate inflammation and joint destruction.  $^{\circ}$ 

### Activated B cells may produce cytokines known to promote inflammation and joint damage in $RA^{\rm 3,4,6,12}$

B cells may be activated to produce cytokines such as TNF- $\alpha$ , IL-6, and lymphotoxin in a variety of ways:

- antigen binding to the B-cell receptor<sup>4,6</sup>
- binding of the costimulatory ligand found on activated T cells, macrophages, and dendritic cells to the costimulatory receptor on B cells<sup>4,6,12</sup>
- exposure of B cells to cytokines produced by other cells<sup>4</sup>

B-cell-produced lymphotoxin may also indirectly perpetuate RA by promoting the formation of new tertiary lymphoid structures in the synovium.

The increased understanding of the potential roles of B cells may provide further insight into the pathogenesis of this systemic autoimmune disease and ultimately change the way we see RA.

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